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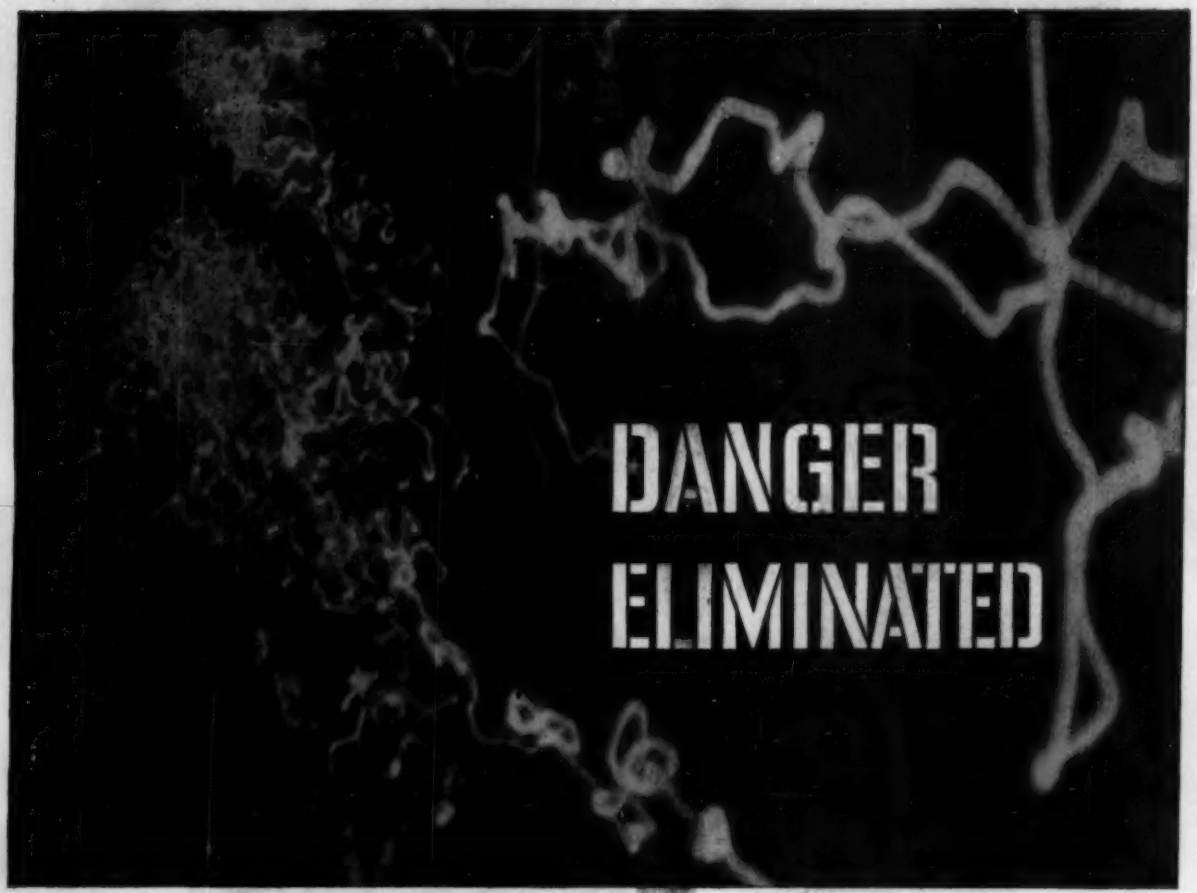

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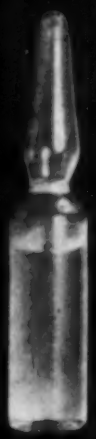

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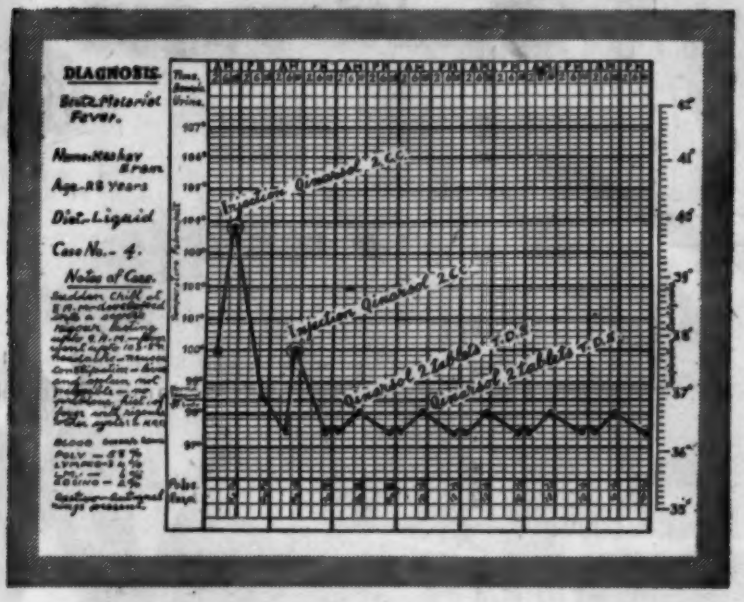

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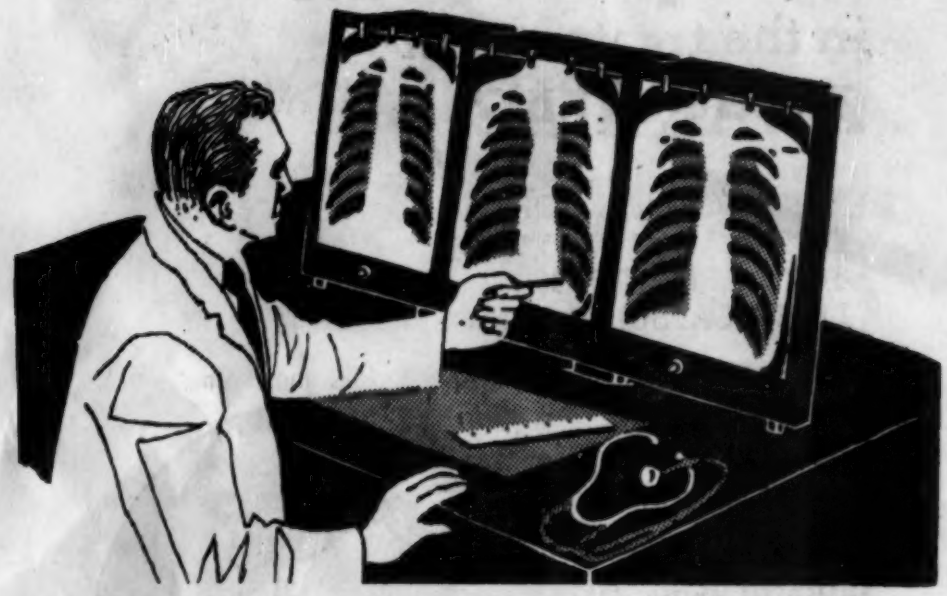

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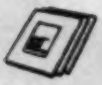

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ORIGINAL ARTICLES

VALUE OF PANCREATIC FUNCTION TESTS IN CASES WITH PAIN IN THE ABDOMEN

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During the past decade notable advances have been made in clarifying the basic mechanism of pancreatic physiology. They have resulted from anatomicophysiologic investigations of gland secretion under normal and abnormal conditions, and from precise physiochemical analysis of the pancreatic juice itself. These data, supplemented by contributions from radiologists and surgeons, when applied to the problems of diagnosis of abdominal pain, help in more exact and reliable diagnosis of pancreatic affections.

The peculiar nature of pancreatic disorder is hardly met with in other fields of pathology. The results of treatment of pancreatic syndromes can be improved by early recognition. A bed-side diagnosis is for the most part a matter of guess work. Biochemical findings are recognised as essential for a correct diagnosis. But such aids are not used as often as they should be.

The importance of correct diagnosis in an acute attack of pancreatitis is obvious, as best results are obtained from conservative medical treatment. Many diagnostic tests for detection of disease of the pancreas have been designed. While considering the limitations of these tests it is well to recall the large functional reserve of the pancreas. A small amount of healthy tissue can produce a normal pancreatic juice and therefore an abnormality may not be demonstrated with any of the existing tests. In a recent review on tests used in the diagnosis of pancreatitis it has been remarked that no pancreatic test is yet pathognomonic (Pollard and Bolt, 1956).

The diseases of the pancreas for which a functional test is necessary, are either inflammatory or neoplastic, and the functional change produced is either pancreatic insufficiency or an obstructive phenomenon. Insufficiency is usually the result of an advanced diffuse lesion, and the obstructive mechanism may result from an acute inflammatory process with minimal fibrotic change or from a neoplasm localised in the head of the pancreas. Therefore, patients with mild inflammatory disease who have not been tested shortly after an attack and those who have only moderate chronic changes may show normal functional response.

Measurements and analysis of pancreatic juice obtained after duodenal intubation have proved tedious in practice and are liable to gross technical error (Dreiling et al, 1954).

Faecal analysis has also proved unreliable except in the late stages of chronic pancreatic disease, because the enzymes of pancreatic and intestinal juices complement each other. Investigations of digestive ferments, amylase and lipase in blood, and diastase in urine have been more helpful to clinicians. The enzymes determination in blood has been found more convenient and reliable than those in the urine.

Wapshaw (1948) found that the diastase and lipase tests were equally satisfactory but their

clinical application was limited to the initial stages of the disease.

Material and Methods

Twenty-five normal healthy persons (mostly medical students) were selected as control. Sixty patients with acute and chronic abdominal pain, admitted to the Gandhi Memorial Hospital, Lucknow, provided the clinical material for the present study.

1. A careful history was recorded and special attention was paid to the past history of intestinal infestation with worms, diabetes mellitus, jaundice and haematemesis or malaria. Habit of alcohol and opium addiction was also enquired into in all the cases.

2. A thorough general and systematic examination was made in all the cases.

3. Routine investigations of stool, urine and blood were made in every case. Special investigations like plain x-ray of abdomen, barium enema and barium meal series, pyelography, cholecystography, gastric analysis and sigmoidoscopy were also done whenever thought necessary, for clinching the diagnosis.

4. Estimations of serum enzymes, amylase and lipase, and diastase in urine were done in all the cases. Fasting blood sugar and serum calcium were done only in cases where serum amylase levels were above normal range.

Note—1. Plasma antithrombin titre test and fat balance studies in stool could not be done due to lack of facilities.
2. Provocative enzyme test has not been included in the plan as it is not of much value (Dreiling *et al*, 1954).
3. Laparotomy was done where required.

The diagnosis arrived at finally in our study of sixty cases is shown in Table 1.

Table 1—Showing Diagnosis of the Cases

Diagnosis	No. of cases
Pancreatitis	2
Acute cholecystitis	3
Carcinoma of gall bladder	1
Renal colic	3
Acute intestinal obstruction	5
Perforated appendix	2
Acute appendicitis	6
Peptic ulcer	8
Chronic colitis	15
Chronic cholecystitis	5
Chronic appendicitis	3
Tubercular abdomen	7
Total ...	60

Techniques of biochemical tests—

1. Serum amylase was done by Somogyi's method (King, 1956).

2. Serum lipase was done by Comfort's method (Todd, Sandord and Wels, 1953).

3. Urinary diastase was estimated by Wohlgemuth method (King, 1956).

4. Blood sugar estimation was done by Folin-Wu method.

5. Serum calcium estimation was done by Clark-Collip method (Todd *et al*, 1953).

Results

In this study twenty-five normal healthy subjects and sixty patients with acute and chronic pain in the abdomen have been observed. Diagnosis in every case was labelled according to one or more of the following criteria:

1. Specific history.
2. Physical signs.
3. Laboratory investigations.
4. Biochemical tests.
5. Radiological examination.
6. Operation.

The results of pancreatic function and other biochemical tests are discussed below:

A. IN NORMAL (CONTROL) SUBJECTS:

(a) *Serum amylase—*

Range: 70 to 180 Somogyi units per 100 ml. of serum.

Average: 138 Somogyi units per 100 ml. of serum.

Values of serum amylase above 200 Somogyi units per 100 ml. of serum are considered pathognomonic of pancreatic disease (Dreiling *et al*, 1954).

(b) *Serum lipase—*

Range: 0·02 to 1·3 ml. of N/20 NaOH/ml. of serum.

Average: 0·55 ml. of N/20 NaOH of serum.

The normal range varies from 0 to 1·5 ml. of N/20 NaOH/ml. of serum (Wapshaw, 1948).

(c) *Urinary diastase—*

Range: 100 to 320 units per 100 ml. of urine.

Average: 180 units per 100 ml. of urine.

But these readings fluctuated much in our cases.

B. IN PATIENTS WITH PAIN IN THE ABDOMEN:

The raised values for serum amylase, serum lipase and urinary diastase in the present series are shown in Table 2.

TABLE 2—SHOWING RAISED VALUES OF SERUM AMYLASE, SERUM LIPASE AND URINARY DIASTASE IN DIFFERENT CLINICAL CONDITIONS

Clinical conditions	No. of cases	Serum amylase			Serum lipase			Urinary diastase		
		200 to 400 units	>400 units	Per cent	<1·5 ml.	>1·5 ml.	Per cent	<400 U	>400 U	Per cent
Recurrent pancreatitis ...	1	—	1	100	—	1	100	—	1	100
Post-operative pancreatitis	1	—	1		—	1		—	1	
Acute cholecystitis ...	3	1	—	33	3	—	—	—	—	—
Carcinoma of gall bladder	1	—	—	—	1	—	—	2	1	33
Renal colic	3	—	—	—	3	—	—	2	Not done in one case	
Acute intestinal obstruction	5	2	—	40	—	—	—	4	1	20
Acute appendicitis ...	6	1	—	37	8	—	—	5	1	37
Perforated appendix ...	2	2	—	—		—	—	—	2	
Peptic ulcer	8	—	—	—	8	—	—	8	—	—
Chronic colitis	15	—	—	—	15	—	—	15	—	—
Chronic cholecystitis ...	5	—	—	—	5	—	—	5	—	—
Chronic appendicitis ...	3	—	—	—	3	—	—	3	—	—
Tubercular abdomen ...	7	—	—	—	7	—	—	7	—	—

N.B. In none of the cases morphia was given before the estimations of enzymes. Serum calcium and fasting blood sugar were done in those cases where serum amylase was found raised. In none of the cases it was low. Fasting blood sugar was also within normal range in all the above cases.

DISCUSSION

Although many cases may have atypical presentation, it is generally taught that pancreatitis should always be considered as a serious possibility in all cases of severe upper abdominal pain, particularly when such episodes are associated with continuous nausea and retching.

For convenience the cases of the present series are grouped under two headings: (1) pancreatic group, and (2) non-pancreatic group.

I. PANCREATIC GROUP:

Not infrequently the serum enzyme tests are found to be unreliable. It has been found raised in certain other acute abdominal conditions such as deranged renal functions, mumps and after the use of morphine, etc. A marked hyperglycaemia may also interfere with the accuracy of the test.

In this series two cases were labelled pancreatitis. One of them was more specifically labelled as a case of post-operative pancreatitis. It is worthwhile to mention briefly the clinical history of these cases.

CASE REPORTS

CASE 1—P.K., 19 years, was admitted with complaints of recurrent attacks of pain in the abdomen with constipation and anorexia for about two years, and constant pain around the umbilicus since a day before the admission. The pain was severe, and was mostly confined to the upper abdomen and radiated to the back. He had 3 or 4 projectile vomitings, and nausea was a constant feature.

On examination: Diffuse tenderness, mostly in the upper part of the abdomen, with rigidity in the epigastrium was present.

The results of investigations are shown in Table 3.

TABLE 3—SHOWING LEVELS OF SERUM AMYLASE, SERUM LIPASE AND URINARY DIASTASE IN CASE 1

Examined on	Serum amylase (Somogyi units/100 ml.)	Serum lipase (ml. of N/20 NaOH/ml.)	Urinary diastase (units/100 ml.)
Day of admission ..	800	5·5	500
2nd day of admission	640	4·0	700
3rd day of admission	530	2·9	400
5th day of admission	160	2·7	280
8th day of admission	80	1·5	100

Fasting blood sugar and serum calcium were done twice in this case, but were within normal range on both occasions. Plain x-ray of abdomen and glucose tolerance curve did not reveal any abnormality. The boy was put on conservative treatment and was discharged.

The follow-up of the case revealed that after a few months, the patient again developed the same trouble. This time he went to Calcutta School of Tropical Medicine, where he was given a purge. An ascaris worm was found in the stool. Since then the patient did not get any further attack, and is now leading a healthy life.

CASE 2—The next case was labelled as a case of post-operative pancreatitis. The brief clinical history is as follows :

S.R.N., 27 years, was admitted with symptoms of peptic ulcer for four years. During the course of this illness there were periods of remissions lasting for about a year or so. A year ago he was operated upon. No ulcer could be found, and the abdomen was closed. He was put on conservative treatment and remained well for another year. He again developed the same trouble with several attacks of haematemesis. He was operated for partial gastrectomy on 4-9-57. During the fourth post-operative day he had severe attacks of bleeding. The wound healed up well. Then suddenly at 6 P.M. on 17-9-57 (i.e. the 13th post-operative day) he felt pain in the right hypochondrium. It was relieved by the passage of flatus and faeces. In the night the pain recurred with nausea. On 20-9-57 (i.e. 16th post-operative day) his condition deteriorated again. A lump was palpable in the right hypochondrium and within a few hours the lump increased in size. A plain x-ray of the abdomen was done, and nothing abnormal was detected. Duodenal leakage was suspected, and laparotomy was done. No leakage or collection of fluid was found. The omentum was grossly oedematous and showed signs of inflammation and necrosis secondary to acute pancreatitis. Three days after the operation the patient expired.

The following investigations were made :

TABLE 4—SHOWING LEVELS OF SERUM AMYLASE, SERUM LIPASE AND URINARY DIASTASE IN CASE 2

Date	Serum amylase (Somogyi unit per 100 ml.)	Serum lipase (ml. of N/20 NaOH per ml.)	Urinary diastase (units per 100 ml.)
21-9-57 ...	510	4·5	480
22-9-57 ...	450	3·0	520
23-9-57 ...	Expired	—	—

Serum calcium and fasting blood sugar were done only once and were found normal.

In both the cases serum amylase level was highest on the 1st and 2nd estimations and gradually declined towards normal on subsequent days. Similarly, serum lipase level was also raised in both the cases. In Case 1, where it was possible to repeat the estimations for several days, serum lipase remained elevated for a longer period of time than serum amylase. In other words, the fall of serum amylase was more sharp than that of serum lipase. Serum amylase level came to normal on the 5th day while serum lipase level came to normal on the 7th or 8th day. Thus, if a case comes 3 or 4 days after the onset of abdominal pain and the clinical features are suggestive of pancreatitis but serum amylase is normal, the estimation of serum lipase may prove helpful.

Values as high as 8,000 units of serum amylase have been reported, but most cases fall within the range of 200 to 800 Somogyi units.

In a series of 25 cases of pancreatitis observed for a period of about 3 years, Anand, Sawhney and Chuttani (1956) accepted levels of serum amylase about 320 Somogyi units per 100 ml. of serum as confirmatory, provided the levels fell to normal after subsidence of the attack. In 3 of their cases where serum amylase figures were not raised at the time of examination, diagnosis was based on operative findings of an enlarged and firm pancreas, scattered fat necrosis and a spastic sphincter. Urinary diastase figures of above 40 units per ml. of urine in their series were regarded as suspicious, although not much relied upon unless serum amylase level also was elevated significantly.

Fat necrosis was induced in the omentum of dogs by Langerhans in 1891 by the injection of pancreatic juice, and he suggested that lipase was the causative agent.

Burnett and Ness (1955) made the diagnosis only at laparotomy in 14 out of a consecutive series of 24 cases of acute pancreatitis. Owing to diagnostic difficulties, the correct diagnosis is frequently made only at laparotomy, usually done for some other conditions like cholecystitis, intestinal obstruction or appendicitis, etc. In a survey of 58 cases of acute pancreatitis treated at Mount Sinai Hospital in the years 1940 to 1949, the diagnosis in each case was corroborated by demonstration of the presence of the disease at operation or at autopsy. Only in 20 out of 58 (39 per cent) cases the diagnosis was made on the basis of serum amylase level ranging from 190 to 575 units.

In both our cases the uninary diastase level was raised but there was much fluctuation in the reading. Chuttani and Anand (1957), considered level of urinary diastase above 400 units per 100 ml. of urine as abnormal.

The urinary diastase level was used for many years for the diagnosis of acute pancreatitis. At present it is employed less often, because the examination of blood serum is of greater diagnostic value.

It has been observed that in normal persons there was wide variation in the amount of diastase present in different specimens of urine taken from the same individual over a 24-hour period, and that these variations did not depend upon the diet he was taking. The urinary diastase value is useful in acute pancreatitis or in the acute stage of chronic relapsing pancreatitis, because the urinary diastase may remain elevated after the serum amylase has returned to normal. When serum amylase is elevated, the urinary diastase

also rises except in diseases of the kidney where normal renal function is impaired.

It will not be out of place to mention that determination of urinary lipase has not been widely used, while a satisfactory method for determination of urinary trypsin is not yet available.

Fasting blood-sugar and glucose tolerance test were within normal range in both the cases.

Serum calcium was also normal in both.

Hence we could not draw any conclusion from the studies of blood sugar and serum calcium estimation in our two cases. Fasting blood sugar and glucose tolerance test will be found to be of value in long standing cases of pancreatitis, where it may reveal a diabetic curve.

II NON-PANCREATIC GROUP :

Considerable attention has now been directed to those conditions other than pancreatic disorders where serum amylase values are also elevated. At times it may cause difficulty or confusion in the diagnosis of pancreatitis. So in every case where the serum amylase level is found elevated, the following conditions must be kept in mind, viz., perforated peptic ulcer, peritonitis, intestinal obstruction, dissecting aneurysm of aorta, mumps, after morphine or pethidine administration, bronchogenic carcinoma, uraemia, pneumonia, after abdominal operations, etc. Many of these conditions can be ruled out when considered in conjunction with the history, clinical findings and results of other investigations.

The determination of serum amylase as the first test to be performed in obscure cases of upper abdominal pain has been rightly emphasised.

Keeping the above objectives in mind, these tests were carried on in non-pancreatic abdominal conditions in order to find out the value of these pancreatic function tests.

In the present series out of 3 cases of acute cholecystitis only 1 case showed serum amylase level above normal, but this was below 300 Somogyi units per 100 ml. of serum, and serum lipase was normal in all the 3 cases. Urinary diastase was also raised in the same single case. Serum calcium and blood sugar were found normal in all the cases. Similarly, serum amylase was raised between 200 to 300 Somogyi units in 2 (40 per cent) out of 5 cases of acute intestinal obstruction. But serum lipase, serum calcium, and blood sugar were normal in all the cases. Urinary diastase was also high in the same two cases.

Serum amylase (below 300 Somogyi units) and uninary diastase levels were found elevated in 1 out of 6 cases of acute appendicitis and in 2 cases of perforated and gangrenous appendix.

No clear explanation is available for the elevated serum amylase level in appendicitis and cholecystitis. Some workers have demonstrated a number of amylase producing organisms in the gut (Baker *et al*, 1950). It is possible that with altered bacterial flora occurring in appendicitis and with other abdominal infections enough bacterial amylase may be absorbed through areas of increased vascularity in the affected region, producing a small increase in serum amylase. Elevated serum enzyme levels in intestinal obstruction may be due to absorption of enzyme through the damaged bowel wall or through the peritoneum. This is supported by the report of hyperlipaemia in intestinal obstruction (Johnson and Bockus, 1940).

Rise in serum amylase level causes rise in urinary diastase level also.

Conclusion

Out of sixty cases of acute and chronic pain in the abdomen observed in this study, only 2 cases (3 per cent) were diagnosed as pancreatitis. Much stress cannot be laid on this low incidence (3 per cent), as the total number of cases is too small.

In those cases in which the history and physical examinations are suggestive of pancreatitis, abnormal values from these tests are of great help to confirm the diagnosis.

In all the early (i.e., seen within two to four days of onset) cases of acute upper abdominal pain where the clinical impression is not suggestive of pancreatitis, these tests will help the clinician to arrive at, or to exclude a diagnosis of pancreatitis.

In cases where serum amylase level is more than 400 units, other biochemical tests are not necessary to establish a diagnosis of pancreatitis.

In cases where serum amylase level is elevated to less than 400 units, the estimation of serum lipase is necessary for establishing a diagnosis of pancreatitis.

The determination of urinary diastase does not prove to be of much value.

In cases of abdominal pain (especially with clinical features not very suggestive of pancreatitis) with serum amylase level less than 300 units and with normal serum lipase level and normal plain x-ray of the abdomen, a diagnosis of pancreatitis should not be made unless it is confirmed on operation or at autopsy.

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THE DIAGNOSIS OF RHEUMATIC FEVER

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For the benefit of the practising physicians the late Dr. T. Dukett Jones, under whom the author had the privilege to work at Boston, suggested certain major and minor criteria as a guide to the diagnosis of rheumatic fever. Although these criteria are now universally employed, their usefulness in India has not been firmly established. The purpose of this communication is to compare the frequency of the various manifestations of rheumatic fever as seen at Boston and Delhi and explore the extent to which these criteria are applicable in the diagnosis of rheumatic fever in India. It is also the purpose of this report to discuss the clinical basis for therapy in rheumatic fever. The data presented here are the author's own observations and no attempt is being made to review or correlate the work of others.

MATERIAL

A clinical study of 603 rheumatic fever patients provides the material for this report. These patients fall into two different groups. The first group of 490 patients were observed at the House of Good Samaritan, Boston, from 1941 to 1956 and the second group of 113 patients were seen at Delhi either in the wards or clinics of Safdarjang and All-India Institute of Medical Sciences Hospitals during the last 3 years. All patients were below the age of 17 years with definite evidence of rheumatic activity. The patients in the first group were in initial attacks but as medical history or documentation was not always reliable in the second group, it is likely that a certain number of patients may have had more than one attack of rheumatic fever.

MAJOR MANIFESTATIONS

The major as well as the minor manifestations of rheumatic fever are given in Table 1.

TABLE 1—SHOWING MAJOR AND MINOR MANIFESTATIONS IN 490 PATIENTS AT BOSTON AND 113 PATIENTS AT DELHI RESPECTIVELY (PERCENTAGEWISE)

Manifestations	Boston per cent	Delhi per cent
MAJOR :		
1. Carditis	53	46
(a) Significant murmurs ...	53	46
(b) Pericarditis	6	8
(c) Congestive heart failure ...	8	15
2. Polyarthritis	58	32
3. Subcutaneous nodules ...	12	3
4. Chorea	19	5
5. Erythema marginatum ...	11	0
MINOR :		
1. Fever	89	93
2. Arthralgia	90	94
3. Prolonged P-R interval in ECG	25	14
4. Increased E.S.R., W.B.C., C-reactive protein	92	94
5. Anti-streptolysin "O" titre of 400 units or more	82	80

Significant murmurs—The earliest and the commonest sign of rheumatic carditis is the appearance of significant murmurs. These murmurs are apical, moderately loud, pansystolic, blowing and well radiating, representing mitral regurgitation or/and early blowing decrescendic diastolic murmurs best heard at the left sternal border indicating aortic regurgitation. Associated with the apical systolic murmur in about a quarter of the patients a mid-diastolic rumbling murmur (Carey-Coomb's murmur) is also audible. Mid-diastolic rumbles do not mean mitral stenosis as they disappear with the disappearance of systolic components. It is important to remember that neither mitral nor aortic stenosis murmurs appear in acute rheumatic carditis. Presence of mitral or aortic regurgitation murmurs may indicate acute carditis or established rheumatic heart disease and it is impossible to distinguish the two by mere auscultation. Nevertheless, the presence of a high-pitched musical component in the systolic phase is indicative of active valvulitis.

In the present series 53 per cent at Boston and 46 per cent at Delhi had significant murmurs. The

commonest murmur was that of mitral regurgitation (70 per cent, 65 per cent), next was mitral and aortic regurgitation murmurs together (23 per cent, 29 per cent) and the least frequent were the murmurs of isolated aortic regurgitation (7 per cent, 6 per cent) in the Boston and Delhi series respectively.

Pericarditis—This cardinal sign recognised by the characteristic superficial scratchy friction rub or pericardial effusion occurred in 6 per cent of cases at Boston and 8 per cent at Delhi. There are certain interesting features of rheumatic pericarditis which deserve special attention, viz., (1) Rheumatic pericarditis is always associated with significant murmurs although the friction rub may be so loud as to obliterate the murmur ; (2) However great the pericardial effusion may be, it never causes tamponade ; (3) Rheumatic pericarditis though years later, may show extensive pericardial calcification at operation or autopsy never progresses into constrictive pericarditis ; (4) Rheumatic pericarditis is often associated with marked leucocytosis up to even 50,000 c.mm.

It is also important to note that presence of pericarditis always denotes rheumatic activity irrespective of the valvular status. In other words, when pericarditis is present either in association with acute rheumatic carditis or in established rheumatic heart disease it indicates activity.

Congestive heart failure—8 per cent of the Boston group and 15 per cent of the Delhi group had congestive heart failure. The higher incidence at Delhi may be due to the fact that majority of Indian patients seek medical aid only when physically greatly disabled and may also be due to the fact as mentioned earlier that all cases seen at Delhi were probably not in their initial attacks. However the commonest cause of congestive heart failure in patients between the ages of 5 and 17, in my experience, is rheumatic carditis both at Boston and Delhi.

Polyarthritis—In more than half and in less than a third objective migratory polyarthritis was present in the two groups respectively. In about half of these patients associated significant murmurs were also present. As a rule the big joints were involved in rheumatic polyarthritis although small joints might also be involved concurrently. When only small joints were involved without other manifestations of rheumatic fever, the diagnosis was more in favour of rheumatoid arthritis.

Subcutaneous nodules—The incidence of subcutaneous nodules was 12 per cent at Boston, whereas only 4 cases (3·5 per cent) were seen at Delhi. These nodules vary in size from a pinhead to that of a large pea, often multiple, commonly found over the elbows, ankles and knees, and sometime better seen than felt, histologically showing Aschoff bodies. Subcutaneous nodules are almost always associated with carditis, and if seen in the absence of rheumatic carditis should raise the suspicion of other forms of collagen disease.

Chorea—This manifestation when present is almost pathognomonic of rheumatic fever if Huntington's chorea can be excluded. In the present series this was observed in 19 per cent at Boston and only in 6 patients (5·3 per cent) at Delhi. Chorea may appear early or late in the course of rheumatic fever and often is precipitated by a mental trauma. Although chorea may appear with other stigmata of rheumatic activity a certain number of patients may only have chorea without any other clinical or laboratory evidence of rheumatic fever. This type of chorea for lack of a better term is described as 'pure' chorea. In the Boston series a third of the cases was of 'pure' type but at Delhi all the six patients had other evidence of rheumatic fever. Recurrence of chorea is quite notorious. I have observed patients where it has recurred after 7 to 8 years but generally it disappears at puberty. It is also of interest to learn that chorea and arthritis never occur simultaneously although it may occur at different times in the same patient. This I believe is Nature's protective mechanism because one may visualise the extreme discomfort a rheumatic fever patient with acute swollen joints may experience if he has violent chorea from movements!

Erythema marginatum—Although this was seen frequently (11 per cent) at Boston, I have not seen this manifestation even in a single Indian patient at Delhi, though the history and description was suggestive of such a rash in a European child. Characteristic features of this rash are nonpruritic, transitory, never appears on the face and has a clear margin encircling an erythematous area—the size varying from an 8-anna piece to the palm of the hand.

Minor Manifestations

The minor manifestations of rheumatic fever (included in Table 1) are the following :

Fever—The peak of rheumatic fever seldom reaches beyond 103°F (orally) and does not last for more than 6 or 7 days. The temperature chart is not characteristic by itself, but swinging or saddle-back type of fevers is not generally seen in acute rheumatic fever. There is a particular type of febrile episode seen in rheumatic fever which is called 'cycling'? Untreated patients show fever for 6-7 days, then remain a febrile for 2-6 weeks and again have another bout of fever and the

cycles are repeated for months together. Febrile bouts are associated with other clinical and laboratory manifestations of rheumatic activity but during afebrile phase there is no evidence of activity. This is present only in 1-2 per cent of rheumatic fever patients.

Arthralgia—History of migratory joint pain seems to be much commoner than objective polyarthritis both at Boston and Delhi. It would appear from the present data that 9 out of 10 patients complained of some joint pain. Joint pain seems to be the earliest manifestation in most of these patients.

P-R interval—Prolongation of P-R interval in electrocardiogram was seen in a quarter of the patients at Delhi. It is possible that if repeated electrocardiograms were taken at Delhi the results would have been more comparable.

Increased ESR, WBC, C-reactive protein—At least two of these three laboratory data were positive in most of the patients in both the groups. A rheumatic carditis patient when in failure may sometime show normal sedimentation rate but will have elevated C-reactive protein. Hence where possible both sedimentation rate and C-reactive protein should be estimated.

Evidence of antecedent streptococcal infection—History of previous sore throat or upper respiratory infection was obtained in 55 per cent of Boston group and only in 30 per cent of Delhi group. However, antistreptolysin 'O' titre of 400 units or more were seen in over 80 per cent in both the groups. Although the presence of a high antistreptolysin titre is helpful but not diagnostic of rheumatic fever, titres below 159 units are useful in excluding acute rheumatic fever.

Diagnosis of Rheumatic Fever in India

From the data presented here it is apparent that erythema marginatum is probably not encountered, and chorea and subcutaneous nodules are rather rare manifestations of rheumatic fever in India. Carditis occurs practically with the same frequency as in Boston, but objective arthritis occurs only in a third of the patients here. As half of the patients with arthritis also have carditis then only two-thirds of the total patients can be diagnosed as rheumatic fever on the basis of Jones's criteria of carditis and arthritis.

On the other hand, arthralgia is quite common and if the combination of joint pain, elevated sedimentation rate or C-reactive protein and ASL 'O' titre of 400 units or more are taken as a triad deserving consideration for the diagnosis of rheumatic fever, then it is expected that the diagnosis will be more accurate. This triad was originally suggested by Dr. Massell of Boston. In the present series 74 per cent at Boston and 70 per cent at Delhi fulfil this triad.

Summary

Although the major and minor criteria as suggested by the late Dr. T. Duckett Jones are useful in the diagnosis of rheumatic fever in Boston, certain modifications are necessary for their application in India. It would appear from this study that the two common criteria for diagnosing rheumatic fever in India would be the appearance of significant murmurs and the presence of a triad of joint pain, elevated sedimentation rate or C-reactive protein and high ASL 'O' titre. It is therefore suggested that the syndrome of joint pain, elevated sedimentation rate or C-reactive protein and high ASL 'O' titre be regarded as the major criterion in the diagnosis of rheumatic fever in India.

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INDICATIONS FOR RESECTION IN PULMONARY TUBERCULOSIS

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Resection of the lung plays a definite role in the treatment of pulmonary tuberculosis. It is becoming increasingly popular not only for removing the diseased components of the lung, but also for its capability to preserve maximum functioning of the lung tissue. Early surgery avoids a prolonged, tedious and expensive stay in the sanatorium, and gives a chance of early rehabilitation for the patient. With the improvement of surgical and anaesthetic techniques and discovery of potent antituberculous drugs, pulmonary resection can now be performed with a wide margin of safety.

In 1943 Churchill and Klopstock published the good results obtained by resectional surgery based on a careful selection of patients. In 1945, Overholt reported 196 cases of resection with a mortality rate of only 24 per cent including poor risk ones. Bickford *et al* (1957) in a series of 1575 cases of pulmonary resection in tuberculosis showed that 87 per cent of their cases were free from evidence of active disease at the end of 5 years. This is indeed a great achievement.

Indications

The indications of resection in pulmonary tuberculosis have recently been standardised. Indications such as thoracoplasty failure, bronchostenosis and bronchiectasis have been universally accepted. It is no longer difficult to categorise the indications for pulmonary resection. In general it should be used in localised unilateral or predominantly unilateral lesions, when simpler measures have failed, or cannot be expected to control the disease. It must be remembered that excisional surgery is always supplementary to the established methods of rest and drug therapy.

The excisional surgery for tuberculosis aims at removing the major offending focus. Pulmonary tuberculosis being a localised manifestation of a generalised disease, it is never hoped that surgery will be able to eradicate completely all tuberculous components in the system. However, with the removal of the major offending focus, it is expected that smaller residual lesions will be taken care of by the general resistance of the patient. Besides, the postoperative use of potent anti-tuberculous drugs, e.g., streptomycin, PAS, INH, contributes to a great extent towards healing.

The proper timing for operation plays an important part in the determination of the result. In the progressive phase of the disease surgery is not a safe procedure. It is indicated only when the disease has been stabilised or tending to be stabilised by the conventional methods of rest and drug therapy. At the same time prolonged use of drug therapy may cause emergence of drug-resistant *M. tuberculosis*, and thereby these valuable drugs fail to give postoperative protection. It is desirable to select suitable patients for surgical intervention as soon as a preliminary trial of drug therapy has failed. Unnecessary delay may bring about a disaster.

The types and sides of operation as done in T. B. Hospital, Kanchrapara, in the period between January 1956 and February 1959 are shown in Table 1 which is followed by a detailed analysis of the lesion and the operation.

Table 1—Analysis of Types of Operation and the Side of the Lung Operated on

Operations	Right side	Left side	Total
Pneumonectomy	6	20	26
Lobectomy	34	15	49
Segmentectomy	15	9	24
Pleurectomy	2	1	3
Lobectomy with segmentectomy	1	1	2

Analysis of Material

During the period between January, 1956 and February, 1959, 104 patients underwent pulmonary resectional operation at T.B. Hospital, Kanchrapara. Prior to resection, all these cases had prolonged rest and drug therapy with streptomycin, PAS, INH, etc. Many of them had extensive trial of collapse therapy, e.g., AP., PP., phrenicolysis and even thoracoplasty. The duration of illness varied from 2 to 10 years.

Table 2—Analysis of the Lesions in Relation to Incidence

Lesions			Incidence		
Types	Sub-types	Sites	Male	Female	Total
A. Endobronchial disease	Tension cavity	RUL	—	1	
		RML & Upp. Lobe-Seg.	—	1	
		RLL	1	—	
		RLL Seg.	1	—	4
	Bronchiectasis	RUL	2	1	
		RUL & Sup. Seg-LL	1	—	
		RLL Seg	—	1	
		LUL	2	—	
		L. Pn.	—	2	
		LLL	—	1	10
	Bronchostenosis	L. Pn.	1	—	
		LUL	1	—	2
B. Solid lesions	Tuberculoma	Rt. Apical & Post Seg.	1	—	1
	Atelectasis	RUL	3	1	
		RLL Seg.	—	1	
		RLL	—	3	
		*Lt. Pn.	1	—	
		LUL	—	2	
		Lt. Apico-post. & anterior Seg.	1	—	12

* Lt. Pn. done due to vascular accident.

TABLE 2—*Contd.*

Lesions			Incidence		
Types	Sub-types	Sites	Male	Female	Total
C. Cavitary lesions	Thick walled cavity	RUL	4	3	
		RLL	1	2	
		LUL Seg.	1	—	
		LLL	2	—	
		LLL Seg.	1	—	
		Bi. lobe.	—	2	
		Lt. Pn.	—	1	17
	Giant cavity	RUL	—	1	
		LUL	1	—	
		Lt. Pn.	—	1	3
D. Residual nodules	Residual nodules	RUL	5	2	
		RUL Seg.	5	1	
		RLL	—	2	
		RLL Seg.	1	—	
		RML Seg.	—	1	
		LUL	—	2	
		LUL Seg.	2	3	
		LLL	3	1	28
E. Destroyed lung or extensive unilateral disease	Destroyed lung or lobe	RUL	1	—	
		Rt. Pn.	1	—	
		Lt. Pn.	2	2	6
	Extensive unilateral disease	Rt. Pn.	2	2	
		Lt. Pn.	4	5	13
F. Miscellaneous group	Thickened pleura	Rt. Pn.	1	1	
		Lt. Pn.	—	1	3
	Disease associated with empyema	RUL Seg.	1	—	
		Lt. Pn.	1	—	
		LUL	—	1	3
G. Thoracoplasty failures	Thoracoplasty failures	RUL	1	—	
		Rt. Pn.	1	—	2
H. Disease situated in the lower part of the lung, i.e., middle and lower lobe	This has been shown in the above classification of pathology				

N.B.—RUL=Right upper lobectomy; RML=Right middle lobectomy; RLL=Right lower lobectomy; LUL=Left upper lobectomy; Rt. Pn.=Right pneumonectomy; Lt. Pn.=Left pneumonectomy; LLL=Left lower lobectomy; Bi-lobe=Bilobectomy; Seg.=Segmentectomy.

A. ENDOBRONCHIAL DISEASES :

We had 16 cases under this heading. The common sequels of endobronchial tuberculosis are tension cavity, bronchiestasis, and bronchostenosis. Active endobronchial disease characterised by submucosal oedema, ulceration, tubercle formation presents a definite contra-indication to any surgical treatment. It is only after the active disease has been quietened down by adequate rest and drug therapy, that surgery can be considered. Collapse therapy has very little role to play in the treatment of endobronchial disease, because of its liability to induce complete collapse in the affected segments and making them the seat of chronic suppuration in future.

B. SOLID LESIONS :

We had 13 cases under this heading. This group comprises tuberculoma and atelectasis. Tuberculoma is the radiological term for an intrapulmonary, round and sharply defined opacity with a minimum diameter of 1 cm. and tuberculous in nature. It is a conglomerate caseous tubercle, usually solitary, which may or may not have bronchial communication. Atelectasis of the lung, lobe or segment represents an area of lung from which air has been completely absorbed, thereby converting it into a solid mass. Atelectasis may result from endobronchial tuberculosis, and it has been considered as a solid lesion because of its inability to collapse further.

C. CAVITARY LESIONS :

We had 20 cases under this heading. These cavities have characteristically thick fibrotic walls. They are either old cavities—the walls of which have become thickened due to gradual fibrosis, or excavation of old caseous foci which have been firmly encapsulated. The thick fibrous walls gradually merge with the adjoining lung tissue. Tuberculous cavities which are over 5 cm. in diameter are termed 'giant' cavities. They are generally situated at the apices of the upper or lower lobe. Their response to collapse therapy is very poor.

D. RESIDUAL NODULES :

We had 28 cases under this heading. These lesions are often found in chest skiagrams. Infiltrations are nodular in appearance, with areas of fibrosis and rarefaction localised in a segment, lobe or lung. In this type, the lung parenchyma is extensively involved. With treatment they retrogress to a certain stage and remain stationary as 'residual' nodules or lesions. These fibrocaseous lesions warrant resection, as many of these lesions reactivate later on.

F. DESTROYED LUNG OR EXTENSIVE UNILATERAL DISEASE :

We had 19 cases under this heading. Destroyed lung or lobe is a condition in which pulmonary tissues show complete or almost complete involvement by cavitation and fibrosis, extensive pneumonic disease or combination of all these conditions. The disease involving all lobes of one lung and not responding to conventional methods, warrant early resection, otherwise the disease may extend to the other healthy lung.

F. MISCELLANEOUS GROUP :

We had 6 cases under this heading. The lung often fails to expand after A.P. treatment. This may be due to the formation of a thick cortex over the visceral surface of the pleura or to underlying collapse, fibrosis, or bronchiectasis. In this state fluid or pus continues to collect in the pleural cavity which further thicken the cortex. The object of treatment is the obliteration or excision of the affected pleural space and expansion of the underlying lung, which can be achieved by decortication, or decortication with pulmonary resection or extirpation of the whole diseased lung with the infected pocket (pleuropneumonectomy).

G. THORACOPLASTY FAILURES :

We had 2 cases under this heading. Sometimes thoracoplasty fails to control the underlying disease or may produce atelectasis or bronchiectatic changes. Persistent positive sputum, recurrent haemoptysis or repeated attacks of respiratory infection indicate excision of the underlying diseased area.

H. DISEASE SITUATED IN THE LOWER PART OF THE LUNG :

The lower lobe disease, particularly the cavity situated in the apical lower segments (so called 'perihilar cavity') and basal segments, responds very poorly to collapse therapy. Its situation very close to the diaphragm and early formation of adhesion may account for this failure. Thoracoplasty may involve the sacrifice of a great deal of functioning lung tissue which amounts to a functional pneumonectomy with uncertain result.

Comments

During the last 15 years, the 'excisional surgery of the lung' for tuberculosis has established itself in the therapeutic programme of pulmonary tuberculosis. With the advent of modern antituberculous drugs, the mortality and morbidity rates of operations have been considerably lowered. As an elective procedure it can now be used with confidence.

The indications for this operation have been considered in this paper. These elective operations are aimed at removing the major offending foci. They should be used only when patients have been stabilised, when the disease has been localised by the conventional rest and drug therapy, and when the patients have developed systemic and local resistance against the disease.

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SEDIMENTATION RATE OF STORED BLOOD

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Erythrocyte sedimentation rate depends on several factors such as fibrinogen and globulin content of blood, size of R.B.C. and rate of rouleaux formation. Melville and Basil (1960) have shown that E.S.R. varies quite significantly with time, i.e., the interval between the hours of collection and examination of blood. The present work was carried out to study the changes in E.S.R. of stored blood, the storage being done under conditions employed in blood banks.

Material and Methods

Blood sample collected from apparently healthy subjects of either sex was mixed with the following anticoagulant-nutrient solution :

Sodium citrate	...	...	300 mg.
Dextrose	...	...	300 mg.
Uric acid	...	...	20 mg.
Distilled water	...	...	100 cc.

The above solution, freshly prepared, was mixed with the blood in the same proportion

TABLE 1—SHOWING RESULTS OF E.S.R. DETERMINATIONS

Sample No.	E.S.R. (mm.) at 24 hrs. interval after collection of blood													
	1st day		2nd day		3rd day		4th day		5th day		6th day		7th day	
	1st hr.	2nd hr.	1st hr.	2nd hr.	1st hr.	2nd hr.	1st hr.	2nd hr.	1st hr.	2nd hr.	1st hr.	2nd hr.	1st hr.	2nd hr.
1 ...	2	4	2	3·5	5	8	1·5	3	0·5	1	×	×	×	×
2 ...	3	6	2	6	3	8	0·5	1	0·5	0·5	×	×	×	×
3 ...	2	5	0·5	1	0·5	1	0	0·5	×	×	×	×	×	×
4 ...	35	67	15	31	14	20	3	7	2	5	×	×	×	×
5 ...	25	55	24	54	20	47	10	20	2·5	3·5	×	×	×	×
6 ...	26	49	5	12	7	19	3	6	5	7	5	7	×	×
7 ...	3	11	3	13	3	10	2	8	2	6	×	×	×	×
8 ...	47	89	56	98	45	91	43	86	22	42	6	12	×	×
9 ...	23	48	23	48	15	32	11	25	2	6	0·5	1	×	×
10 ...	14	26	18	43	20	40	13	36	11	28	9	20	7	13

(blood : anticoagulant :: 4 : 1) as prescribed for blood transfusion. The E.S.R. was tested, within 15 minutes of dilution, by the Westergren's method. Readings were taken at the end of the 1st hour and at the end of the 2nd hour.

The blood in a test tube was stored in the refrigerator. After 24 hours the blood was taken out, very gently and thoroughly mixed and allowed to attain the room temperature. Then the E.S.R. was again estimated. This was repeated every day up to 5-7 days.

RESULTS

Details of the findings from 10 normal subjects are shown in Table 1.

DISCUSSIONS

The aim of this work was to find out how long the blood could be stored in the blood bank without any appreciable change in E.S.R. In six of the ten samples (60 per cent) there was no change in sedimentation rate after 24 hours of storage. In three cases there was a retardation and in one case there was acceleration of E.S.R. On the 3rd day of storage there was not much change in the sedimentation rate. But on the 4th day there was a dramatic retardation of E.S.R. in almost all the cases. The cause of this retardation is not definitely known and intensive investigation is necessary for finding out the cause which might lie either in the plasma or in the corpuscles.

SUMMARY

Storage of blood under the same conditions as in blood bank for more than 72 hours alters the interrelationship between the plasma and the red blood cells, which is manifested by a marked retardation in the sedimentation rate of the blood.

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JAUNDICE IN INFANCY AND CHILDHOOD

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Jaundice in the newborn is a problem which concerns its immediate life as well as its future development. Factors responsible for the production of jaundice are: (1) excessive breakdown of red cells; (2) hepatocellular or parenchymal damage; and (3) an anatomical or organic biliary obstruction. The causes of jaundice in infancy in such conditions as haemolytic disease of the newborn, sepsis, syphilis and congenital atresia of the bile ducts are fairly well understood. There is still a major group of cases with uncertain aetiology which are labelled 'physiological' jaundice and an ill-defined group designated as 'hepatitis'. The differential diagnosis of jaundice in the newborn often presents some difficulty, as the haemolytic type is frequently complicated by obstructive elements, and not infrequently a jaundice associated with parenchymatous type of damage to the liver will appear clinically as a case of complete obstruction.

Unlike in adults, liver function tests are of doubtful value at this age. The most valuable tests are those concerned with bile pigment changes. Bile pigment is consistently present in the urine of jaundiced infants. Urobilinogen is also present in haemolytic disease of the newborn and neonatal hepatitis; and its occasional presence in total congenital atresia of the bile duct is interesting but unexplained. The presence of bile pigments in the stools is an important aid in distinguishing between parenchymatous and obstructive jaundice in the neonatal period, as variable amounts may be present in the faeces in haemolytic disease and virus hepatitis.

The bilirubin level is helpful in diagnosing the development of kernicterus. High values indicate the necessity of exchange transfusion. The level reaches a peak and comes down with recovery in haemolytic disease of the newborn, whereas in atresia of the bile duct the level rises slowly and continuously. The other tests like serum flocculation tests, serum cholesterol determinations, and serum alkaline phosphatase level, are either of limited or of no diagnostic value. Liver biopsy is not very helpful.

Some of the principal causes of jaundice in the newborn (Nicholson, 1956) are as follows:

A. WHERE JAUNDICE APPEARS AT BIRTH OR WITHIN THE FIRST WEEK OF LIFE:

1. *'Physiological' jaundice*—(a) In full-time infant. (b) In premature infant.
2. *Haemolytic disease of the newborn* (erythroblastosis foetalis)—(a) Due to Rh incompatibility. (b) Due to ABO group incompatibility. (c) Due to other blood group incompatibility.
3. *Infection*—(a) Neonatal hepatitis. (b) Congenital syphilis. (c) Toxoplasmosis. (d) Cytomegalic inclusion cell disease.

B. WHERE JAUNDICE IS PRESENT IN THE INFANT AFTER THE FIRST WEEK OF LIFE:

1. *Prolonged obstructive jaundice*—(a) Atresia of bile ducts: (i) Extrahepatic ducts. (ii) Intrahepatic ducts. (b) Inspissated bile syndrome of erythroblastosis foetalis. (c) Neonatal hepatitis: (i) Viral— ?Homologous serum hepatitis— ?Infectious hepatitis— ?Other viruses. (ii) Toxic— ?Bacterial toxins.
2. *Prolonged 'physiological' jaundice.*
3. *Galactosaemia.*
4. *Congenital familial non-haemolytic jaundice with kernicterus—Constitutional hyperbilirubinaemia*—probably inherited as a recessive characteristic. Elevation of indirectly acting serum bilirubin (very uncommon).
5. *Haemolytic anaemias*—(a) Acholuric jaundice. (b) Cooley's anaemia. (c) Sickle-cell anaemia. (d) Hereditary non-spherocytic haemolytic disease.

Some of the important aspects of jaundice in infancy are considered below:

Physiological Jaundice

Jaundice seldom appears before the 2nd day of life and usually disappears by the 10th. It commonly occurs in normal infants. There is no associated splenomegaly and the condition resolves with no untoward sequelae.

Physiological jaundice was thought to be due to haemolysis of excess of red blood cells which were no longer required by the newborn, and gave rise to hyperbilirubinaemia. This theory seemed unlikely because such increased haemolysis would lead to increased faecal stercobilinogen, but the reverse actually occurs. Those with the highest serum bilirubin levels, excrete the least pigment in the stools—suggesting deficient hepatic secretion rather than haemolysis. It has been suggested that foetal erythrocytes are more susceptible to haemolysis, than the adult ones. But survival studies have shown that placental erythrocytes are not more fragile than those of the adult. It seems probable that the liver of the newborn is functionally immature and is unable to excrete bilirubin efficiently. This is supported by grossly abnormal bromsulphalein tests, confirming diminished excreting capacity for pigments. It would explain the more severe jaundice of premature infant. Physiological jaundice is present in 50 per cent of premature infants, and lasts up to 4 weeks and may be intense. No treatment except fluids is required.

The jaundice may be pathological in premature children and may cause kernicterus. Hottinger (1958) of Basle found 7 per cent of kernicterus among deaths of premature babies in 1922-29; 7·3 per cent in 1950-53; and 22 per cent during 1954-56. The increase is significant. Kernicterus is no longer considered as a complication only of Rh incompatibility or other blood group incompatibilities. It is a disease of premature babies also. Physiological icterus appears less frequently with increasing maturity of liver. The so-called 'physiological icterus' appears earlier and is more intense in infants weighing less than 1500 g. (approximately 3·3 lb.). Rising concentration of serum bilirubin means increasing danger of kernicterus at levels exceeding 20 mg. per cent. It is

necessary to perform exchange transfusion in premature children where the bilirubin content is very high (over 20 mg. per cent). In spite of the exchange transfusion, a kernicterus may arise if bilirubin accumulated in the tissues is diffused into the blood, raising the serum bilirubin level. This calls for a second exchange transfusion.

Haemolytic Disease of the Newborn

This disease includes three syndromes as manifestation of the same disease entity, viz.,

1. Congenital haemolytic anaemia,
2. Icterus gravis neonatorum, and
3. Congenital oedema (hydrops foetalis).

Clinically the disease may manifest itself as a mild undiagnosed anaemia due to a severe icterus neonatorum, or the next severe form of hydrops and intra-uterine death. The fundamental cause of the disease is incompatibility between the foetus and the mother with respect to one of the blood group factors. Antigen in foetal red cells may provoke the development of maternal antibodies which passing into foetal circulation, leading to haemolysis of foetal red blood corpuscles. The commonest incompatibility is in the Rhesus blood factor. About 15 per cent of mothers are Rh-negative and are therefore liable to produce antibodies if the foetus is Rh-positive. The blood of an Rh-positive person is capable of being agglutinated by the serum of a sensitised Rh-negative individual and the responsible Rh-positive antigen is often referred to as 'D' and its antibody as 'anti-D'. The Rh blood group like other blood groups is inherited as a Mendelian factor—the genes being carried by a chromosome pair different from those carrying all other known blood groups, each chromosome being contributed by a parent. Thus a person's constitution (or genotype) in this respect may be DD (Rh-positive homozygous), Dd (Rh-positive heterozygous), or dd (Rh-negative homozygous). In Great Britain 83 per cent of all persons are Rh-positive and 17 per cent Rh-negative. Among the former the heterozygous type is probably somewhat more numerous than the homozygous. This is significant, because the spermatozoon of a homozygous father will carry gene D, and if he is married to an Rh-negative woman who becomes sensitised, all babies will run a considerable risk. On the other hand, approximately half the children may escape when the father is heterozygous.

An Rh-negative woman married to an Rh-positive man is in danger of becoming sensitised to Rh antigen, but only 1 in 20 of such women does develop sensitisation. This seldom happens with the first pregnancy after marriage unless the mother had previously received injections or transfusion of Rh-positive blood or has had previous pregnancies (including abortions and stillbirths). Immunisation by blood injection or transfusion is a much greater menace to the Rh-negative woman than pregnancy. It is thought that about 50 per cent of Rh-negative persons develop antibodies following a single transfusion or even a very small subcutaneous or intramuscular injection of Rh-positive blood. In western countries the incidence of haemolytic disease is 1 in 200 of all pregnancies. Its incidence is lower in eastern countries. Every pregnant woman should have her Rh-group examined. If she is Rh-negative her blood should be examined for antibodies and the test repeated 6 weeks before the expected date of confinement. The presence of antibodies in the mother's serum indicates that the foetus, if Rh-positive, is under considerable risk of developing haemolytic disease of the newborn. The attachment of the Rh-antibodies to the erythrocytes of an Rh-positive may be shown by direct Coombs' test performed upon the blood taken from the child within one week of birth, or preferably from the umbilical cord at birth.

Clinically this jaundice appears within an hour of birth or so, develops rapidly, and deepens from hour to hour. The infant becomes drowsy and anaemic, and the liver and the spleen are enlarged. Petechiae and even free haemorrhages may develop, especially when jaundice is present at birth. What other features point to the diagnosis of haemolytic diseases?

Prenatal evidence—History of jaundice at birth in a previous pregnancy, or known case like this in a previous infant in the family is important. History of Rh-negative mother having received a blood transfusion or intramuscular injections of blood even remotely in the past, or history of one or more miscarriages in a mother known to be Rh-negative is important.

Postnatal evidence—Amniotic fluid may be yellow coloured, and the foetal end of the cord may be stained. The placenta is rather large. Jaundice appearing within an hour or so after birth and deepening rapidly. Liver and spleen are palpable. Reticulocytosis, nucleated R.B.C. and macrocytosis may be present in stained films and Hb. estimations reveal progressive anaemia. Cord blood haemoglobin is more reliable. Coombs' test is positive.

Subsequent course of the disease—The main hazard of the disease, apart from the damage from haemorrhage and anaemia, is the high serum bilirubin level. It is the most important complication.

Kernicterus

This is a condition where marked staining of the basal ganglia and subthalamic nuclei occurs with marked necrosis. It may occur when the serum bilirubin level rises above 6 mg. per 100 ml.

The serum bilirubin measures and kernicterus—With peak bilirubin, 0 to 5 mg. per 100 ml. there is no kernicterus. With peak bilirubin 6 to 15 mg. per 100 ml. the incidence of kernicterus is 3 per cent. With peak bilirubin 16 to 30 mg. per 100 ml. the incidence of kernicterus is 18 per cent. Where bilirubin level is over 30 mg. per 100 ml. the incidence of kernicterus is about 50 per cent. This grave nuclear involvement is a frequent complication of jaundice of prematurity and haemolytic disease of the newborn. It may also complicate other forms of jaundice, especially neonatal hepatitis.

Symptoms usually appear between the fourth and eighth day of birth. The jaundiced child refuses to suck or vomits, becomes restless or lethargic. The infant develops stiff neck, and retraction of head, which may proceed to opisthotonus. There is stiffness of limbs, squint, twitchings, convulsions, and high-pitch cry. Death may supervene rapidly in 12 hours and 70 per cent of affected infants die within 7 days of onset of the disease. Those who survive, develop permanent neurological sequelae like mental defect, cerebral palsies, athetosis, deafness, green staining and ridging of the deciduous teeth, unless they die of intercurrent infection.

The chief aim in treatment is to avoid the development of kernicterus. Early exchange transfusion can reduce the incidence of kernicterus. Where indicated, exchange transfusion must be performed within 6 hours of birth, and may have to be repeated. Premature children with jaundice and those in whom haemolytic disease of the newborn is suspected or has developed, must be hospitalised.

When the cord haemoglobin concentration exceeds 17·5 g. per 100 ml., the infant is very unlikely to get kernicterus. Infants with cord Hb. concentrations between 15·5 and 17·5 per 100 ml. should be carefully examined at intervals not exceeding 8 hours during the first 24 hours of life, and any suspicion of the onset of jaundice within this period should be considered as an indication for exchange transfusion. Assuming that the direct antiglobin is positive, indications for immediate exchange transfusion should be: (a) birth weight 6 lb. or less, or infant born 3 weeks or more before the expected day of delivery (irrespective of any other finding), (b) history of the mother having previously given birth to a similarly affected infant (irrespective of any other findings), and (c) cord Hb. concentrations below 16·5 g. per 100 ml. (Mollison and Cutbush, 1954).

An infant whose bilirubin concentration is rising towards 20 mg. per 100 ml. despite a first exchange transfusion, is a very good case for receiving a second transfusion. Sometimes even a third is required.

Inspissated Bile Syndrome

The inspissated bile syndrome is a type of obstructive jaundice caused by a less severe complication of haemolytic disease of the newborn—the jaundice being attributed to the inspissated bile blocking the bile canaliculi. This should be suspected clinically when jaundice due to Rh incompatibility persists unduly long and is of obstructive type. Persistence of jaundice over 10 days after exchange transfusion or over 3 weeks after onset in untreated patients is very suggestive. It may persist for 15 weeks, but eventual recovery is usually complete.

ABO Group Incompatibility

Jaundice occurs within 24 hours after birth, and is usually mild but sometimes marked. It affects the first birth as well as later births. Spontaneous recovery is usual. Other blood group factors are uncommon causes of haemolytic diseases.

Neonatal Infections

Infections or general sepsis and septicaemia from extraneous sources, usually umbilicus, skin, gastrointestinal tract, etc., are causative factors—the infections being due to *Staphylococcus aureus, E.coli*, and streptococcus. Jaundice usually occurs on the 5th day or later. The child refuses feeds. Diarrhoea, collapse and dehydration with slight pyrexia may develop. The child rapidly deteriorates. These infections are of less importance since the advent of the antibiotics.

Congenital toxoplasmosis—The protozoan causing this disease may be transmitted to the foetus from an unsuspected maternal infection. Jaundice may be present within a few hours of birth, and may be associated with hepatosplenomegaly and encephalomyelitis and followed by hydrocephalus, microcephaly, retinochoroiditis and intracerebral calcification. The spinal fluid may be xanthochromic. The diagnosis may be confirmed by isolation of causative organisms in mice inoculated with blood of C.S.F. or by demonstrating neutralising complement fixing or cytoplasm modifying antibodies in the serum. Jaundice is due to direct hepatic involvement and haemolysis.

Herpes simplex—The liver may be involved in the course of fulminating virusaemia which may be contracted at birth from herpes simplex infection of maternal genital mucosa. Jaundice is due to viral involvement of the liver. Characteristic herpetic lesions may involve the mucosa of the infant.

Congenital syphilis—Visceral involvement is common in foetal infection and the foetal liver is the particular site in which a large number of *Treponema pallidum* can be found, leading to fine pericellular cirrhosis.

Neonatal Viral Hepatitis

The virus of serum hepatitis is small and is capable of traversing the placenta and infecting the foetus. Human transmission studies have indicated the presence of infective agent in the serum of mother and infant and transplacental spread seems the only portal of entry of the virus to the infant (Stokes *et al*, 1951).

The agent transmitted by this route is probably the serum hepatitis virus rather than the infective hepatitis virus because the foetus is protected from the latter by gamma globulins in the mother's blood. True infective hepatitis is rarely transmitted by the pregnant mother to the foetus unless the disease is contracted late in pregnancy. The mother may be a healthy carrier of the serum hepatitis virus and transmit it in successive pregnancies. In one such family three infants in succession were affected (Dible *et al*, 1954).

The disease may be a cause of stillbirth or the affected infant may die soon after birth before jaundice had time to develop. Clinically obvious is the type in which icterus of various grades is present during the first 12 days of life. Recovery may occur within a few weeks or the disease may last weeks, months or years, passing through the stages of chronic hepatitis to cirrhosis. The diagnosis from congenital obliteration of the bile duct may be very difficult. Exploratory laparotomy is indicated when the infant is 3 to 4 months old. With liver biopsy the condition is found similar to adult hepatitis, with liver necrosis, fibrosis, bile duct proliferation, and attempts at liver cell regeneration.

Prognosis in infancy, as distinct from later childhood, is poor. Survivors may well be forerunners of the obscure cirrhosis for which no cause can be found (Dibble *et al*, 1954).

Treatment—Adequate protein, fat soluble vitamins and high caloric intake are recommended.

Prolonged Obstructive Jaundice

Congenital atresia of the bile duct—Any malformation of extrahepatic biliary system may occur, e.g., absence of part or complete absence of the whole system. There may be late canalisation of apparent fibrous biliary ducts.

Pressure from outside causing obstruction—e.g., choledochal cyst, annular pancreas, etc.

Obstructive jaundice with normal biliary tree—(1) inspissated bile syndrome ; (2) atresia of intrahepatic interlobular bile ducts (developmental dysplasia).

Congenital Atresia of Bile Duct

Here jaundice is the outstanding symptom. It may be present at birth, but is usually not seen until 2 or 3 weeks later. It may present as physiological jaundice which does not clear up. Jaundice in this case is progressive without remission. The infant is deeply icteric or of deep bronzed hue. Stools are pale or clay coloured, or even white meconium may be present. Urine is deeply bile stained with no urobilinogen. The liver is large, firm and smooth and later cirrhosis and ascites develop. Appetite and nutritional status are good for many months. These children seldom live beyond 2 years. McDonald Jr. (1957) has pointed out that 60 per cent of these infants with obstructive jaundice are born with atresia of bile duct, and 25 per cent with intrahepatic disease, especially hepatitis. The difficulty in distinguishing the two cases commonly leads to delay in operation for up to 6 months until hepatitis has been excluded. He has found that early biopsy of the liver, through a small incision under local or cyclopropane analgesia will provide an accurate diagnosis within one or two months of birth and permit early exploration of biliary atresia before the liver is irrevocabaly damaged.

Galactosaemia

Obstructive type of jaundice usually appears after the first week of life. It presents with history of infant failing to thrive, anorexia, large liver, and possibly cataract. It is an inborn error of metabolism affecting the enzyme system concerned with conversion of galactose to glucose. The mode of hepatic damage is uncertain. It is controlled by galactose-free diet.

Vitamin K and kernicterus (Gellis, 1957-58)—During the last couple of years reports have appeared of kernicterus in premature infants who had received high doses of vitamin K analogue (synkavit). The mechanism of synkavit in increasing blood bilirubin in premature infants is uncertain, although it may be a cause of haemolysis or may interfere with the capacity of the liver to handle bilirubin. The effect of vitamin K dosage on plasma bilirubin levels in premature infants is

reported by Bound and Telfer (1956) who compared two groups of premature infants. The only significant variable was the dosage of vitamin K analogue, synkavit. Plasma bilirubin levels in venous blood were estimated on the 5th day. Among 55 infants who received 30 mg. synkavit (10 mg./day for 3 days), the mean 5th day bilirubin level was 15·4 mg./100 ml., and 21 had levels over 18 mg./100 ml. By contrast, in 51 infants receiving only 1 mg. synkavit, the mean plasma bilirubin level on the 5th day was 9·7 mg./100 ml. and only 2 had levels over 18 mg./100 ml. Both these differences are significant. Two infants in high dosage group died of kernicterus, and 3 had transient symptoms of the condition. In no infant of the low dosage group did kernicterus develop.

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PRACTITIONERS' SERIES

CONVULSIONS IN CHILDREN

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Few symptoms are more terrifying to witness than are 'fits' or episodic convulsions. The phenomenon is a dramatic one, appearing abruptly and often violently. The condition is embarassing to the parents, other witnesses, and even to the attending doctor. Unfortunately convulsions are very common in children, especially of the younger age groups. The incidence of convulsions in relation to other childhood diseases is estimated to be one in twenty. This is roughly 15 times more frequent than in adults where it is estimated to be 1 in 300. This has been attributed to the immaturity of the central nervous system from lack of myelin, high water content, more rapid metabolism and perhaps some other chemical peculiarities so that minor infections like injuries, emotional and metabolic upsets, to which the adults are fairly immune, readily provoke convulsions in young children. Electro-encephalographic patterns in children show immature cerebral rhythm with higher waves and slower frequency.

SIGNIFICANCE OF THE TERM 'CONVULSION'

Convulsions constitute only a symptom, arising from some cause whether organic or functional and to find out the cause, certain investigations may be required. However, in some cases, the cause may remain undefined in spite of the laboratory aids. Nevertheless, a very thorough and carefully taken history of events preceding and accompanying the symptom is indispensable. Detailed information on the perinatal history, past and family history is important. In the present history, the onset of the illness, course of the fever, headache, vomiting, dizziness, muscle cramps, blood in the urine with oliguria, dietary deficiency, poisoning of some kind, head injury or haemorrhagic tendency, exposure to communicable disease, temper tantrum and the existence of a known hereditary predisposition would help to unfold the underlying nature of the disorder responsible for the seizure. Likewise a detailed physical examination, noting especially the conditions of the anterior fontanelle and the pupils, and the presence of such signs as abnormalities of the muscle tone, apnoea, cyanosis, bradycardia, temperature record, neck rigidity would be rewarding. The causes of convulsions in children are indeed numerous and varied. By itself, the fit or seizure is just an incidental symptom of secondary importance in the ultimate prognosis which naturally depends on the primary cause. By contrast, in 'idiopathic epilepsy', seizure is, of course, the main event.

AETIOLOGY

Convulsions may be due to functional or organic causes and may merely herald the onset of an acute illness or be a terminal sign. Some of the important causes are listed below:

A. *Local causes* (with involvement of brain)—

1. Congenital defects, e.g., agenesis, porencephaly, microcephaly, hydrocephaly, tuberose sclerosis, Sturge-Weber syndrome.

2. Traumatic—natal and postnatal. The nature of the intracranial lesion may vary considerably from contusion, compression, oedema, intracranial haemorrhage including subdural haematoma, etc.

Another form of brain trauma is chemical, especially 'anoxia' resulting from complicated obstetrics and anaesthesia.

3. Infections, e.g., meningitis, encephalitis, cerebral abscess, sinus infection, cerebral malaria, tetanus, toxoplasmosis.

4. Degenerative conditions, e.g., Tay-Sach's disease, Schilder's disease.

5. Neoplasms including granulomas, e.g., tuberculoma.

6. Haemorrhage resulting from various bleeding disorders.

B. *General causes*—

1. Infections with rise of temperature, from any cause, e.g., acute tonsillitis and other upper respiratory tract infections, pneumonias, acute pyelonephritis. These convulsions are called 'febrile convulsions.'

2. Haemolytic disease of the newborn complicated by kernicterus.

3. Anoxia—as in Stokes-Adams syndrome, coughing bouts of pertussis, 'breath holding spells'—and severe cyanotic heart disease.

4. Drugs, e.g., lead, camphor, strychnine, caffeine picrotoxin, cocaine, ergot, metrazol, epinephrine, etc. etc.

5. Metabolic disturbances, e.g., hypoglycaemia. hypocalcaemia (tetany), alkalosis, uraemia as in acute glomerulonephritis, dehydration with electrolyte imbalance, especially hypernatraemia, and also in hypotonic dehydration.

6. Nutritional (pyridoxine deficiency).

C. *Epilepsy*—

It may be emphasised that conditions such as constipation, phimosis, teething, thread and roundworm infestations are not primary aetiological factors. It is the increased inherent convulsive tendency that manifests itself as convulsion by trivial causes like constipation, etc. In fact, when a child is brought with fits, it must be thoroughly assessed whether the exciting cause was sufficiently potent or of an intensity to produce the seizure in a normal child or that there is some abnormality of central nervous system, predisposing it to go into convulsions with either no obvious cause or very weak stimulus.

The above list is by no means complete and it can be further expanded. It, however, accounts for most of the important causes.

From the prognostic point of view, seizures may be spontaneous or idiopathic and secondary or provoked, due to infections, inflammations, trauma, metabolic upsets, etc. The incidence of convulsions caused by acute infections is higher in the younger age groups as against the incidence of chronic recurrent fits in older subjects. The various aetiological factors operate with varying frequency at different age groups, as further elucidated in the order given below:

1. Neonatal period (0 to 4 weeks)—Birth injury, congenital defects of brain, acute infections (local and general), metabolic disorders, anoxia, etc.

2. Early infancy (1 month to 4 months)—Acute infections (general and local), residue of birth injury, congenital defects, etc.

3. 4 months to 2 years—Acute infections (general and local), tetany, residue of birth injury, congenital defects, etc.

4. Preschool period (2 to 6 years)—Brain infections, acute infections, epilepsy, residue of birth injury and congenital defects, tetany.

5. School age (6 to 14 years)—Epilepsy, brain infections, acute infections, residue of birth injury and congenital defects.

It is, thus, clear that whereas any of the above causes may operate at any group, the relative frequency of these causes varies differently when considered chronologically. Generally speaking, birth injury and congenital defects of the brain are more important causes soon after birth and early infancy while infections, general or local, account for the majority of the cases in the later age periods.

Investigations

The investigations that will help to discover the cause are:

Total and differential leucocyte counts to exclude pyogenic infection and malarial parasite; urine examination for urinary infection and for evidence of acute glomerulonephritis; cerebrospinal fluid (complete examination both chemical and microscopic) to determine the various types of meningitis and encephalitis, tuberculin (Mantoux) reaction; roentgen studies of the skull—for any calcification, increased intracranial tension, suture widening, craniostenosis, etc.; fasting blood sugar for hypoglycaemia; serum calcium for hypocalcaemia; blood urea for renal dysfunction and V.D.R.L., Kahn or W.R. tests, electroencephalograms and other selective investigations such as air studies (pneumoencephalography) and cerebral angiography, would be indicated from the history and physical examination.

Some of the 'not-well-understood' Causes

1. *Febrile convulsions*—These constitute a definite entity without any demonstrable lesion of the central nervous system. Their incidence has been variously reported from 26·2 to 51·9 per cent. They are particularly common in the age-group 6 months to 4 years. They are called 'febrile convulsions' because of their association with raised

temperature. The role of body temperature seems to be related to two main factors, namely, age of the child and the manner of the rise or the degree of sharpness of the rise in temperature. Some children get the convulsions only when the fever is over 103°F. while in others, these convulsions appear when the temperature is sharply rising. Generally speaking, they are more likely to occur with a sudden rise of temperature and in a majority of cases occur with a body temperature range of 101°-104°F. The relation of convulsions to fever is important. If the convulsions occur at the onset of fever, when the temperature is 'shooting', they are likely to be 'febrile'. If, on the other hand, they occur during the course of a pyrexial illness, the chances are that these are due to some complication such as meningitis or an abscess. Convulsions which last as long as half an hour or more are not likely to be febrile convulsions. The fact that these convulsions do not appear in all children with raised temperature points to a predisposition or 'soil' which is readily fired by the raised temperature. This predisposition and the young age are two main features which determine the possibility of convulsions and hence as age advances, the incidence of febrile convulsions lessen and older children seem to withstand the rise of temperature with relative immunity. The susceptibility of the young is also confirmed experimentally in kittens which manifest no seizure when grown into adult cats. How does fever cause seizures is difficult to explain. Various explanations given are retention of fluid, increase in aminoacids, temporary alkalosis from hyperventilation, cerebral anoxia, etc. The prognosis of these febrile convulsions should be guarded. A positive family history of epilepsy adversely affects the prognosis. In the affected children with a positive family history, the risk of future epilepsy is great. The duration and character of the fit is important. Convulsive seizures of focal distribution lasting longer than 15 minutes or accompanied by deep cyanosis are ominous. Besides these qualifications of the fit, if these seizures recur three or more times, they may be taken as potentially epileptic. On the other hand, if there is no history of epilepsy in the family and the child had no convulsions before except one at the onset of an acute febrile episode, the single convulsion may be regarded as innocent. The prognosis is also adversely affected if the seizures are severe or recur with even mild infection.

2. *Breath-holding attacks*—These are not related to epilepsy, or any organic cause. These are crying spells arising in young children, mostly 6 to 18 months of age and usually disappear by 3 years. Very occasionally, they are reported in older children. The episode begins with the crying of the child in temper tantrums. The child ends his cry with the breath held in expiration. The severity of clinical manifestations depend upon the breath-held interval. Cyanosis develops in 5 seconds, limpness in 10-15 seconds and convulsions in 20 seconds, to be followed by sleep or limpness in some cases. Clinically, these spells differ from epilepsy in the sequence of their progress and in the absence of incontinence of urine and faeces. The fit occurs during crying or may follow sudden pain, or when something is denied to the child and he wants to have his own way. The prognosis is good and the attacks are not fatal and parents need be fully assured on this matter. Proper management of the child in relation to his environment is important. A significant point is to let the child come to his own and not to fuss over his condition. Splashing the face with cold water, holding the child upside down may abort the attack in some cases. Sedatives and tranquilisers may help to soothe the irritable child.

3. *Kernicterus* is usually a sequela of haemolytic disease of the newborn—with an incidence of 8-30 per cent of the so-called 'icterus gravis' cases. Convulsions occur during the active phase in the neonatal period between the 3rd and 12th days and in some cases, later on, in the chronic phase when the child presents with athetosis, mental retardation, deafness, cirrhosis and green teeth. Kernicterus which is popularly termed as 'nuclear jaundice' is believed to occur in children with a serum bilirubin of 20 mg. per cent or over. Antigen antibody reaction is put forward as the pathogenesis. Prematurity is an important predisposing factor.

4. *Spasmophilia* (tetany of the newborn) may manifest as twitchings or convulsions instead of the characteristic spasms of the hands and feet. In many cases, there is laryngeal spasm—laryngismus stridulus. The condition of tetany in the newborn is probably due to the functional immaturity of the parathyroid glands and kidneys in the newborn from hyperactivity of the maternal parathyroid leading to relative hypofunction of the infant's parathyroid with resultant tetany. In later infancy, hypocalcaemic fits seem to occur more in artificially fed infants, possibly due to the high content (6 times more in cow's than in human milk) of phosphates precipitating calcium. Tetany with rickets is, however, not very common and is met with only occasionally. Serum calcium in rickets is usually normal—the disturbance is in the serum phosphorus and alkaline phosphatase levels, the former is reduced and the latter is elevated.

5. *Epilepsy*—The term 'epilepsy' implies a chronic nervous disorder characterised by recurrent paroxysmal attacks or fits of disturbed consciousness with or without convulsions or any other symptoms or signs, resulting from disturbances in the electrical activity of the brain. This symptom or symptom complex may result from many predisposing and precipitating causes such as individual make up, personality reaction, excitation, physiological disturbances as superhydration, alkalosis, anoxaemia, etc., and lastly in hereditary or innate tendency. The relative importance of these factors is variable and for purposes of treatment, needs proper aetiologic and nosologic evaluation. When epilepsy is present without any apparently demonstrable structural abnormalities of the brain, its blood vessels and membranes, it is called primary or essential or idiopathic or cryptogenic to distinguish it from organic or symptomatic epilepsy.

TABLE 1

Clinical type	Localisation
SOMATIC MOTOR	
1. Generalised seizure (*grand mal*)	Complete motor
2. Jacksonian seizure (local motor)	Pre-Rolandic gyrus
3. Masticatory seizure ...	Lower Rolandic
4. Simple adversive seizure ...	Frontal
5. Tonic postural seizure (decerebrate, opisthotonic) ...	Brain stem
SOMATIC SENSORY (AURAS)	
6. Somatosensory seizure (tingling, numbness, sense of movement, etc.)	Post-Rolandic gyrus
7. Visual seizure (lights, darkness, colours)	Occipital
8. Auditory seizure (buzzing, drumming, etc.)	Temporal
9. Vertiginous seizure (sense of dizziness)	Temporal
10. Olfactory seizure ('uncinate fit')	Infratemporal
VISCERAL	
11. Autonomic seizure (viscerosensory and visceromotor, e.g., pupillary, circulatory and gastrointestinal references) ...	Diencephalic
PSYCHIC	
12. Dreamy state seizure (illusional and hallucinatory seizures) ...	Temporal
13. *Petit mal* (lapses, absences) ...	?
14. Automatism (psychomotor attacks or epileptic 'equivalents')	?
15. Psychotic states (secondary) ...	?

The various well recognised clinical forms and their probable localisation as summarised by Penfield and Erickson (quoted from Brennemann's Practice of Pediatrics) are shown in Table 1.

The typical attack in *grand mal* consists of prodromal symptoms or aura, the motor reactions, consisting of the initial tonic and succeeding clonic spasms with loss of consciousness and the post-convulsive phase and in some cases automatism. When the attacks appear in rapid succession, without return of consciousness, the condition is called 'status epilepticus'.

Petit mal is a very intriguing disorder and may show as transient attacks of unconsciousness described as fainting, lapses or dizzy spells or loss of posture control (akinetic or Salam type) or as myoclonic with sudden jerky movements of flexor muscles of arm—throwing the object held in the arm violently.

Pyknolepsy, narcolepsy and cataplexy are rather very rare forms. In pyknolepsy the spells may vary in number, i.e., from several to as many as hundred or more a day. There are no muscular spasms or falling to the ground during seizures and no mental confusion or drowsiness follows. This is really a variant of *petit mal*.

Psychomotor epilepsy presents fundamentally as some transient behaviour disturbances, with personality changes such as dreamy state, visuo-auditory hallucination. There is impairment of consciousness and the patient may become destructive or aggressive or become morose and apathetic or go into temper tantrums or become loquacious or run away, jump up and down indicating total amnesia or disorientation. The electro-encephalogram is characteristic showing prolonged periods of dysrhythmia with local spikes and occasional brief periods of normal rhythms intervening.

The somatic sensory components consist of tingling, numbness, loss of feeling, or visual with light disturbance, flashes of light, star and ball images, uncinate form and unpleasant gustatory sensation.

The term 'partial epilepsy' used in preference to the old term 'focal epilepsy' implies local discharge in some part of the brain and depending upon its intensity and spread, the clinical picture may vary from major or minor fits to localised spasm, sensory disturbance, dreamy states and mastication movements.

The popular term, focal or Jacksonian epilepsy usually includes motor unilateral fit, indicating the origin in the motor cortex.

Epilepsy in general, usually occurs in children above the age of 3 years and the incidence rises

with age. Many cases of recurrent febrile convulsions in which fits recurred repeatedly in early life, pass on to epilepsy—the fits occurring without fever.

The role of heredity in epilepsy is important. How does the family history of epilepsy influence the prognosis of the individual case and what is the incidence of epilepsy amongst the relatives are difficult and not fully settled questions to answer. The incidence of epilepsy in monozygotic twins is estimated at 56 per cent, dizygotics 10·4 per cent, offspring 5·9 per cent, siblings 3·7 per cent, nephews and nieces 1·2 per cent and general population 0·4 per cent. It may, thus, be said that heredity predisposes to epilepsy through inherited structure and inherited function. However, it does not follow that epilepsy is directly transmitted. A tendency to it is found in children whose ascendants have been of psychopathic stock, suffering from migraine, epilepsy, mental disorders, etc.

6. *Intracranial neoplasms* are not uncommon in children and the common tumours are tuberculomas, astrocytoma, medulloblastoma, craniopharyngioma, papilloma of choroid plexus. Unlike adults, infratentorial tumours are more frequent in children than supratentorial—ratio approximately 2 : 1. In adults, the ratio is reversed. It follows, therefore, that convulsions in children from brain tumours occur mostly from the rise of the intracranial pressure and seldom from local irritation.

7. *Intracranial infections*—Convulsions from intracranial infections such as encephalitis and meningitis need no comment and examination of the cerebrospinal fluid is the only means of ascertaining the conditions. Fits commonly occur during the course of tuberculous meningitis and rarely also during pertussis, measles and typhoid encephalopathy.

8. *Pyridoxine deficiency convulsions*—Since the experimental production of convulsions in rats and pigs by feeding them on a diet lacking in pyridoxine, there has accumulated ample evidence that pyridoxine deficiency causes convulsions, apathy, failure to gain weight and anaemia and that administration of pyridoxine corrects the condition. Human milk has a high level of pyridoxine. The incidence of pyridoxine deficiency convulsions is, therefore, present mostly in artificially fed children on defatted cow's milk.

Treatment

Naturally, treatment of convulsions means the treatment of the cause responsible for convulsions and therefore every attempt must be made to find out the cause. However, since the symptom itself is damaging and alarming, it should be considered as an emergency and controlled as quickly as possible with one or the other drug. It may be true that some seizures stop spontaneously in an hour or so but in most cases, they may last longer and continue to recur and in cases where they last longer, particularly in a child under the age of 3 years, they may cause irreversible cerebral changes, possibly from anoxia and oedema.

Drug therapy :

The choice of drugs is discussed briefly under 2 headings ; namely drugs controlling convulsions in general and drugs for controlling epilepsies.

(a) *Drugs for the control of convulsions*—Bromides, chloral hydras, phenobarbital, paraldehyde, aether anaesthesia by the open mask method are the main drugs used. Paraldehyde is a drug of low toxicity and does not depress the respiratory centre unless given in very large and toxic doses. It needs no sterilisation. Deep intramuscular injection is safely given and the incidence of abscess formation is very rare. The dose is 1 c.c. per 10 lb. of body weight. If the attack is not readily controlled in half an hour, half the initial dose may be safely repeated. Intramuscular paraldehyde is quite an effective and safe procedure. Rectal route is also occasionally employed but there is no advantage in it and, besides absorption is undependable.

Barbiturates are very useful and easy to be administered. They are, however, respiratory depressants. Phenobarbital is the drug of choice especially as a treatment in between two seizures. The approximate doses according to age are : infants under 1 year, ¼ grain ; 1-5 years, ¼-½ grain ; 5-10 years, ½-1 grain ; and 10-14 years, ¾-1½ grain. During the acute attack intramuscular barbiturate ½-1 grain under one year, and 1-2 grain in 2-5 years age group is both safe and satisfactory method of controlling the fits. The dose may need to be repeated 4-hourly, until the patient is free from convulsions for 24 hours. Sodium seconal capsule given rectally restores tranquillity and produces sleep in 15 minutes or so. Overdosage with barbiturates may cause incoordination and prolonged sleep or profound anaesthesia with paralysis of respiration. When taken for long periods, they have cumulative action.

Chloral hydras is another safe drug in the dosage of 1 grain per 1 year of age. The total daily dose may be 3-5 grains up to 6 months and 5-10 grains above 6 months.

If after one to two hours of the above mentioned active and adequate anticonvulsant treatment,

the seizures still continue, more active steps as discussed under epilepsies, such as intravenous paraldehyde dissolved in normal saline or aether inhalation may be required. Aether anaesthesia is quick, safe and usually effective in some apparently resistant cases. Only a few drops suffice. The aim of anticonvulsant therapy is to control the seizure as quickly as possible with as small a dose of a drug, with the least harmful side-effects. It is often possible to control the seizures completely with doses that do not produce unpleasant side-effects. In intractable cases, a combination of drugs is often better than a single one. As the fit is being controlled symptomatically with one drug or another, the cause must be searched for and remedied.

TABLE 2

Drug	Dosage	Indications	Side-effects
Barbiturates	0·03-0·09 g. (½-1½ gr.)	*Grand mal*	Ataxia, slurred speech, confusion, depression, drowsiness, skin rashes.
Hydantoins:		*Grand mal* Psychomotor	Measly rash, fever, lymphadenopathy.
Epinutin or Dilantin or Phenytoin Sodium	0·06-0·4 g. (1-6 gr.)		Gum hypertrophy (especially when septic but not with Mesontoin)
Mesontoin	—do—		Tremors, ataxia, diplopia, nystagmus, psychosis.
Mebaral	0·06-0·18 g. (1-3 gr.)	*Grand mal*	
Mysolin (I.C.I.)	0·25-0·5 g. 1-6 times daily	*Grand mal* Psychomotor	Drowsiness, slurred speech, accommodation upsets, less toxic.
Milontin	0·5-1·5 g.	*Grand mal*	
Phenurone	0·25-0·5 g. 1-4 times daily	*Grand mal* Psychomotor	Hepato-toxic, jaundice, vomiting, skin rashes, blood dyscrasias.
Diones: Tridione Paradione (Abbot) Malidione (British Schering)	0·3 g. capsule 1-4 times daily	*Petit mal*	Agranulocytosis, skin rashes, drowsiness, photophobia, glare phenomenon.

(b) *Drugs in the treatment of epilepsy*—The choice of drug with dosage, indication and side-effects for the common types of epilepsies has been indicated (Table 2).

Bromides have now practically been replaced by phenobarbital and other anti-convulsants, because of their unpleasant taste, interference with appetite and the tendency to chromic bromism. They are, however used in combination with other drugs such as chloral hydras. Phenobarbital is the most widely used single anti-epileptic drug which produces satisfactory results. Fortunately, it is not only effective but is easy to take and has few side-effects. The drug must be taken regularly, beginning with small doses and gradually increasing the dose to full therapeutic efficiency. Hydantoins are usually combined with barbiturates—the latter is given at night and the former during the day.

Tridione and allied drugs are of specific value in the various types of *petit mal* epilepsy in which other drugs are ineffective.

Amongst the other drugs occasionally used in *petit mal* epilepsy are benzedruic (amphetamine), caffeine, ephedrine and diomax. Magnesium and calcium salts, diuretics, cathartics, snake venoms, iodides, parathyroid extracts, borotartrates, etc., have now been relegated to a very small place in therapy and are only very occasionally used.

The importance of maintaining anticonvulsant therapy long after the occurrence of the last seizure needs full appreciation. Two years are hardly long enough in most cases. Sudden withdrawal may precipitate 'status epilepticus'.

TREATMENT OF STATUS EPILEPTICUS:

This presents as an emergency requiring urgent and heroic treatment. Half measures are useless. Paraldehyde, I.M., alone or supplemented with luminal sodium, I.M. or I.V., often breaks the seizure cycle. Aether or chloroform inhalation to the stage of light anaesthesia, with a face mask are also a safe and satisfactory method of getting the seizures under control speedily. Pentothal sodium is another effective and safe procedure. Amytal or chloral or ammonium bromide per rectum, large dosage of urea by mouth and spinal fluid drainage have also been recommended. This anticonvulsant therapy must be continued even after consciousness is restored. In addition to the heroic therapeutic measures with the various anticonvulsant drugs, intravenous fluids (of course with moderation), antibiotic therapy and adequate hydrotherapy are essential.

HISTORY OF SURGERY

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Bombay

In this Boeing age we have broken the sound barrier and transportation at an incredibly swift rate is now possible. Today I hope to break the time barrier and take you through a journey extending over 45,000 years in the space of this article.

The question is often asked, 'When did surgery begin?' Surgery began when an ancient man met with an accident and there was bleeding.

The earliest human remains belong to a man who lived about 45,000 years ago in Iraq. These now lie in the famous and fabolous Smithsonian Museum at Washington, U.S.A. This skeleton was uncovered by a Smithsonian archaeologist Dr. Ralph Solecki in 1957. These remains have been ascribed to Shanidar Man who succeeded the Neanderthal Man. Dr. T. Dale Stewart, an international authority on physical anthropology, states that his Shanidar Man was about 40 years old and suffered from arthritis. What is interesting is that his right arm was amputated just above the elbow. Dr. Stewart calls it the earliest evidence of human surgery on record. The mural paintings in the cave of La Tene in France depict many hands with fingers amputated especially the little finger. The amputations presented in the mural paintings of La Tene may have been done for purposes of magic, ritual sacrifice, punishment or cure of a disease. These are about 6,000 to 10,000 years old. An ancient Peruvian pottery shows the figure of a man with a leg amputated at tibiotarsal junction, holding in his right hand a pointed cap-like structure, which is probably a device to be adjusted to the amputated limb to facilitate walking. It is not true to say that dead men tell no tales. The bones speak a language of their own if you can decipher it. What may we not hear on the day of resurrection!

Trepanning or trephining of skull was performed 10,000 years ago in Neolithic times especially in Western Europe and Bohemia. At a later date this operation was also performed in Bolivia, Peru, North America, Mexico and Central America. In the Smithsonian Museum there is a collection of 20,000 skulls from Peru and other places. About a hundred of these skulls show evidence of trepanation. These holes in the skull show signs of healing. Some skulls show several trephine holes of different dates, some more healed than others. Lucas Championniere says that this operation is still performed by some primitive tribes. He talked to one of the trepanists who belonged to a family of trepanists and who himself had undergone the operation four times. According to him the operation was done for relief of pain in the head. Broca surmised that this operation was done in prehistoric times for epilepsy and for driving out evil spirits from the head.

Circumcision seems to be an operation of great antiquity. In an ancient Egyptian temple this operation is illustrated in some wall paintings. Egyptian surgery was not of a high order.

In the Ebero Papyrus there is a description of an operation for cataract. The Egyptian ophthalmic surgeons had a high reputation and practised in the court of the great Persian King Cyrus. Incidentally Persia will be celebrating the 2,500th anniversary of Cyrus's death next year.

India had a brilliant exponent of surgery in the person of Sushruta, who probably flourished a couple of centuries before Christ. He describes over 120 surgical instruments and tells us how to take care of them. Splints and bandages (14 varieties) have been described. His appreciation of surgery will remain a classic. "Surgery is the first and highest division of the healing art, least liable to fallacy, pure in itself, perpetual in its applicability, the worthy product of heaven, the sure source of fame on earth".

Surgery in Greece under Hippocrates was also of a standard comparable to India. Five hundred years after the death of Hippocrates, Galen flourished in Rome. Galen taught laboratory methods through animal experimentation. He was the first to employ ligatures to arteries; but not to end vessels in amputation. This our 16th-century Paré did. Galen even gave the name of the shop where the ligatures could be purchased. Antyllus, Roman surgeon, treated an aneurysm by tying the artery above and below the sac and evacuating its contents. But to stop bleeding, only boiling oil or red-hot cautery was employed.

Then followed the dark ages and all further progress stopped. Surgery remained in the hands of barbers and itinerant specialists such as the lithotomists. The most important phase in the development of medicine came when universities were established in Europe. Till then one could learn medicine by working as an apprentice to another established doctor. Now the ancillary sciences began to grow rapidly and it was not possible for one man to know all these new developments. Now that medicine was being taught at the university level, the status of medicine rose;

but until John Hunter came on the scene (1728-93) surgery remained a mere craft. He raised it to the status of a science.

John Hunter was a prodigious worker and was content with few hours of sleep. From breakfast till 12 noon he saw patients in his office. After twelve, he would see nobody. Then he went for his calls and hospital duties and returned home at 4 p.m. and had an early dinner. After that he slept for an hour. From 5 30 p.m. to midnight he worked in his laboratory and made notes of his observations. He went to bed at two in the morning. He was indeed a cheerful companion for his wife! He stressed the importance of embryology and comparative anatomy. He anticipated any number of modern discoveries. His famous operation, Hunter's operation for popliteal aneurysm, was the direct application of his observations on the growth of antlers in the deer. He caught a buck and tied the external carotid on one side. He observed that the antler became cold to touch on the operated side. To his surpise two weeks later the antler was warm again. So he killed the animal and found the ligature intact, but found the establishment of collateral circulation. He concluded that under the stimulus of necessity the smaller vessels were capable of an increase in size. A case of popliteal artery was admitted under him in St. George's Hospital. Should he try the old Antyllus operation of double ligature and evacuation of clot or should he tie the artery high enough for collateral circulation to form? He decided to tie the femoral artery in the thigh. Right down to our times this operation has persisted and is known as Hunter's operation. He collected a large number of specimens illustrating human anatomy, comparative anatomy, physiology and pathology. The total number of specimens amounted to 13,682 which cost him £125,000. In 1800 this collection was acquired by the Royal College of Surgeons of London. Hunter raised not only the status of a surgeon but also that of a pathologist. The pathologist who opened a dead body was no longer a ghoul but a real student and scientist. As a colleague of his said after Hunter's death, "John Hunter made us gentlemen".

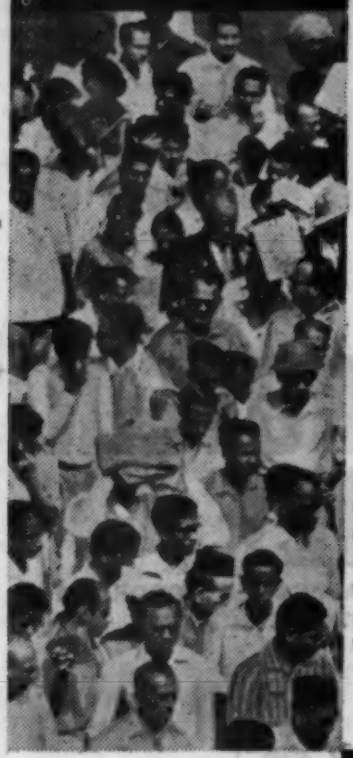
There were now three factors which hampered the progress of surgery—pain, sepsis and shock. Benjamin Brodie (of Brodie's abscess fame) stated emphatically in 1847 that some means to make the operations painless was one of the greatest needs of mankind. However he said it was a vain wish and would never be accomplished. Even as he said so a ship was sailing from America bearing the tidings of the greatest discovery in the surgical world—the discovery of ether as an anaesthetic. Mr. Horace Wells, a dentist in Boston, witnessed an exhibition of laughing gas and noticed that his friend Cooley who had voluntered to inhale the gas, was dancing and laughing, even after knocking his shin hard against the edge of a bench. When he questioned Cooley, if he suffered any pain, Cooley denied it. When Wells pulled up Cooley's trousers, he saw blood trickling from a big gash. It was then, that Cooley became conscious of pain. Wells immediately conceived the idea that a tooth could be pulled out painlessly under the influence of laughing gas. He offered himself as a patient and had his tooth pulled out under gas and felt no pain. After several such successful extractions, he presented himself before Dr. Warren in January 1845 and wanted to prove his technique of painless extraction. Dr. Warren said to his class that there was no case for him to show to Mr. Wells but if some one in the audience wanted to have his tooth pulled out painlessly he could avail himself of Mr. Well's services. A stoutish individual came forward and gas was administered. The man's head fell forward and his face looked bluish in colour. But the man woke up just when the tooth was being extracted. He let out a tremendous scream and the students shouted 'Humbug', 'Humbug' and poor Wells had had to beat a hasty retreat. Wells persevered and soon became well known for painless dentistry.

Mr. W. Morton, one-time partner of Wells, now took up the idea and on 30th September, 1846, visited the laboratory of Professor Jackson for certain information about nitrous oxide gas. Jackson having no nitrous oxide gas on hand suggested a trial of sulphuric ether. Morton experimented with ether and with the help of Jackson devised an inhaler with a glass mouthpiece. Meeting with success he approached Dr. Warren of Massachusetts General Hospital and offered to demonstrate his method. On 16th October, 1846, he was invited to give ether to a patient of Dr. Warren. Morton had to make some last-minute adjustments so he was not there in time. Dr. Warren said sarcastically, 'As Dr. Morton has not arrived, I presume he is otherwise engaged'.

Morton arrived a few seconds later and apologised to Dr. Warren for being late. A little impatiently Dr. Warren said, "Well Sir, your patient is ready'. Morton administered aether and in a few minutes said, 'Sir, your patient is ready'.

Dr. Warren with a few swift strokes of the knife removed the tumour from the face of his patient and ligated the vessels and stitched up the wound. The patient did not turn a hair, and Dr. Warren remembered the words 'Humbug', 'Humbug', shouted by the students when poor Wells had failed only a year ago. In a voice choking with emotion he said: 'Gentlemen this is no humbug',

Facts and Figures



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and there were tears in his eyes. The clock struck ten and a new page was turned in the history of surgery on November 18, 1846. Henceforth the great dread of pain was removed from surgery. Bigelow announced the discovery to the world in a Boston medical journal. Liston of London used aether as an anaesthetic in December, 1846, Syme of Edinburgh in 1847 and Pirogoff of Russia in 1848.

Before the advent of anaesthesia, speed counted for everything. Thus Liston could do an amputation in 28 seconds. Chiselden the great lithotomist at St. Thomas' Hospital could remove a stone from the urinary bladder by perineal section so rapidly that you would miss the entire operation if you happened to cough at the time.

With varying fortunes surgeons practised surgery. Some abdominal operations were successful. Many others failed. The cause of failure remained a mystery. In the cases that failed, the clinical picture that preceded death was the same, whether the patient died after a laparotomy or after a compound fracture. The patient had fever with rigors, delirium and the wound had a foul odour. The same picture presented itself when a woman died after a confinement. For some unexplained reason the uterus was not sutured after performing a caesarian section. It was wrongly argued that by virtue of its inherent contractile power the uterus would close automatically. But no one could realise that the open uterus acted as a ready-made channel for sepsis to enter the peritoneal cavity *via* the vagina. The mortality after caesarian was close to 100 per cent when Porro of Italy while doing a caesarian could not control the bleeding and in sheer desperation he tied a wire round the cervix and controlled the haemorrhage, removed the body of the uterus and stitched up the abdominal wall. That patient lived and Porro's operation was adopted and the mortality dropped by half. Porro by tying the wire round the cervix had shut off the communication between the peritoneal cavity and the vagina and the mortality dropped by half. Every caesarian was converted into a Porro's operation.

Dr. Ignaz Philipp Semmelweis in the obstetric hospital at Vienna observed that mortality following childbirth was several times higher in the ward where medical students conducted the cases as compared with the ward where cases were conducted by nurses. He traced this discrepancy to the fact that the students came directly from their dissections to do the obstetric cases. He thought that some contagion from the dissection room, carried by the students on their hands, was the responsible factor. He therefore compelled his students to wash their hands thoroughly in a basin of chlorine water before entering the wards. The mortality in his ward dropped to the same low figure as in the nurses' ward. Semmelweis was descredited by his seniors who persecuted him and drove him out of Vienna bringing in political pressure. Semmelweis settled down in Budapest and wrote his book on *'The Aetiology, the Concept and the Prophylaxis of Puerperal Fever.* At the 36th Congress of German scientists in 1861, he could only find one supporter for his theory. The Great Virchow opposed it because it did not fit in with his concept of cellular pathology. Thus an observation of far-reaching importance made by Semmelweis was passed over by the profession.

Joseph Lister observed the horrible results of compound fractures. These results were never seen in cases of simple fracture. He was debating these points with others when Prof. Anderson, a chemist at Glasgow, gave him a copy of Pasteur's paper (1864) on fermentation of wines. Pasteur showed that wine exposed to air became sour. When the flask containing wine was first heated and its mouth sealed so that no air could enter, the wine would not ferment for months. Lister saw in this, the explanation of the difference in the behaviour of the simple and compound fractures. The compound fracture was like the flask of wine exposed to air. He therefore argued that it must be through the air that contamination of the wound must be taking place. His problem was threefold: (1) to prevent ferments getting into wound, (2) to kill the ferments should they find entry, and (3) to prevent further contamination.

Compound fractures gave out a foul stench. A visiting surgeon from Carlisle drew Lister's attention to the wonderful effect of carbolic acid in removing the stench from a sewer in Carlisle. Thus Lister and carbolic acid came together. Lister had to wait 10 months before he could get a case of compound fracture. Today you have not to wait so long, thanks to the motor age. Lister tried the effect of carbolic acid in his compound fracture case in March 1865. The first case was a failure. On 12th August, 1865, he had his second case. He washed the wound with carbolic lotion and changed dressings every day having previously soaked them in carbolic lotion. The case did extremely well. There was no foul-smelling wound. There was no temperature. He thus ushered in the era of antiseptic surgery.

His results following this technique were so good that he published his first article on the subject in *the Lancet* of 16th March 1867. However Lister's views were not easily accepted when he read his paper on the antis-ptic principle of the practice of surgery at the British Association meeting at Dublin on August 9, 1867. At the end

of 1869 Lister received good news from Germany that Prof. Karl Thiersch, Professor of surgery at Leipzig, had tried his system with gratifying results. Those who decried Lister, forgot that Lister not only used carbolic acid but made many radical changes in the preoperative procedures. The hands were to be washed before an operation. Instruments and dressing had to be soaked in dilute carbolic lotion before being used. A fresh apron had to be worn at the time of the operation. He introduced the use of carbolised catgut in 1868.

It was left to Robert Koch to demonstrate what Pasteur had postulated and Lister had suspected—namely, actual micro-organisms were responsible for sepsis. Lister said in one of his adrresses: "You must be able to see with your mind's eye the septic ferments as distinctly as we see flies. If you can, you will be properly on guard against them". In his makeshift, small room, at the German town of Wollstein, Koch made his world-famous discoveries of the bacilli of anthrax, tuberculosis and cholera. He identified the organisms responsible for sepsis. These discoveries revolutionised both medicine and surgery. Again Virchow who had crushed Semmelweis now spoke against Koch. But this time Koch triumphed.

Lister had assumed that germs came from the air. Von Bergman's assistants, Lange and Schimmelbusch tried to culture germs from the air. They found that the germ content of air was low, whereas it was very high in the dust from the floor. Soiled dressings, contaminated hands and instruments were potent sources for sepsis. Koch showed that live steam killed bacteria and spores. Schimmelbusch showed that exposing instruments, bandages, dressings, ligatures to live steam would ensure absolute freedom from microbes. Lister saw his mistake, and gave up his carbolic acid technique. This proves that Lister was not a bigot but a great man and a true scientist worthy of our greatest respect. In 1886 Schimmelbusch introduced sterilisation by steam under pressure and the era of aseptic surgery was ushered in.

Dr. William Halsted who had been to Germany and seen all these developments in the causation and eradication of sepsis was appointed Professor of Surgery at the recently founded Hopkins Medical School. Halsted was always smartly dressed. He wore a top hat, he bought his shirts in Paris and what is more he sent them back to Paris to be washed and ironed. He had one Miss Caroline Hampton as his chief operating room nurse. She was not only highly efficient as a threatre nurse but she alone could make coffee as Halsted would like it. In the Winter of 1889 the skin of Caroline's hands showed signs of eczema arising out of the use of mercury perchloride lotion. Something should be done for Caroline. Halsted experimented and finally hit upon the idea of rubber gloves. Thick rubber gloves were in use in the post-mortem room. Halsted got Goodyear Rubber Co. to make gloves which looked like a second skin. These could be sterilised by steam. Caroline was cured. Halsted married her on 4th June, 1890. She naturally left the threatre. But surgical gloves remained and became indispensable. Dr. Bloodgood when he put on the gloves for the first time remarked, 'What is sauce for the goose is sauce for the gander.'

The last improvement in surgical attire came when in 1911 Willian Hunter of Charing Cross Hospital introduced and advocated the use of gauze masks, to be worn over the surgeon's face. Till about 1911 surgical gloves and gauze masks did not come to the J. J. Hospital.

Long before 1886 when aseptic surgery came to be practised bold surgeons had entered the abdominal cavity, sometimes with amazing results.

In 1602 Florian Mathias, a barber surgeon at Prague, opened the stomach of a professional knife swallower, who had literally swallowed a knife. The story goes that magnets were applied to draw the tip of the knife to the abdominal wall. When the tip could actually be felt, Florian the surgeon cut upon it, removed the knife, stitched up the stomach and the man lived.

Thirty-three years later (1635) surgeons of Königsberg opened another stomach to remove a knife. It had been swallowed by a young farm hand who had been tickling his throat with the knife to induce vomiting after a drinking bout.

Ephraim McDowell, a Kentucky doctor, successfully removed a huge ovarian tumour in 1809 under the most dramatic conditions. The operation was performed on a kitchen table and villagers stood outside the cottage ready to hang him if he should fail.

After the birth of aseptic surgery, abdominal operations were performed in large numbers. Billroth won immortal fame by the performance of his first successful gastrectomy in 1882.

Yet it is strange that the great assassin of the abdomen, the appendix, had not received any serious attention from the surgeon. As early as 1642 Saracenus described an abscess which broke through the abdominal wall on the right side. A hundred years later, Mestivier, a French physician, opened up such an abscess. The patient died. Mestivier dissected the body and found a burst appendix. In 1812 the English physician Parkin-

son dissecting the body of a child having a similar history was the first to point out the possible connection between the appendix and general peritonitis. About 1822 a French physician Mellier by his postmortem studies pointed out that it was the appendix that was at fault in all cases of socalled typhlitis or perityphlitis. Dupuytren, the great surgeon, pooh-poohed the idea of Mellier.

Many a young man coming up the ladder of fame is knocked down by a man coming down the ladder. Dupuytren was coming down the ladder. On April 15, 1848, Henry Hannoch a young surgeon (39) at Charing Cross Hospital, saw a woman with perityphlitis. She was treated on conservative lines with administration of morphine. On April 17, her condition was desperate. Hannoch felt a hard non-fluctuating mass in the right iliac fossa. As her condition became worse, Hannoch in sheer desperation cut down on the swelling under chloroform and the woman finally recovered. He read a paper before the London Medical Association on 25th September, 1848, and recommended surgical interference even before an abscess formed. His words were not heeded.

In 1886 Reginald Heber Fitz, Professor of Pathological Anatomy at Harvard Medical School, delivered a sensational lecture on appendicitis and emphatically stated that all cases of inflammation of the caecum had their origin in the appendix. He coined the word appendicitis. He advocated removal of the appendix as early as possible. The surgeons resented. After all who was a pathologist to suggest an operation to a surgeon. On April 27, 1887 Dr. George Thomas Morton (the son of G. Morton who made history by giving ether on 16th October, 1846) first successfully removed a suppurating appendix. Eleven months later he successfully removed an inflamed appendix. One year later Charles McBurney in New York reported on seven appendicectomies with six recoveries.

John Benjamin Murphy of Cook County Hospital, Chicago, had studied Fitz's publications on appendicitis carefully. A young workman was under his care in the Cook County Hospital, for a broken leg. Suddenly on 2nd March, 1889, the patient with the fracture complained of pain in the right side of the abdomen. Murphy diagnosed it as appendicitis and operated within eight hours of the starting of the pain. The ease with which the operation and the recovery went off led Murphy to believe that he had hit on a revolutionary principle in the treatment of an inflamed appendix. 'An inflamed appendix must be removed as early as possible'. By November, 1889, he had operated on 100 such early cases. Whenever he operated within 24 hours of the attack there were no complications. Murphy enunciated the triad of symptoms—pain, vomiting and a small rise in temperature, as diagnostic of appendicitis. The cult of early operation was soon adopted by the U.S.A. surgeons, but Europe and England adhered to the old trend of cooking an appendix till it formed an abscess.

Frederick Treves, a young surgeon at the London Hospital, removed a chronically affected appendix in the interval between two attacks on 29th June 1888. However he did not become a convert to Murphy's creed. He operated upon cases of recurrent appendicitis after the attack had subsided.

This was the state of affairs until the 24th of June, 1902, when Treves operated upon King Edward VII. On 13th June, 1902, the King did not feel well while reviewing a parade at Aldershot. On the morning of the 14th he complained of pain in the abdomen. He attended a military tattoo but by midnight the pain got worse and he vomited heavily. Sir Francis Laking his personal physician suspecting perityphlitis, called Sir Thomas Barlow the surgeon in consultation on the 15th. He concurred with the diagnosis. In the afternoon of the 15th the King had chills and fever. On the 16th the condition improved and the King was brought down to Windsor Castle. A considerable quantity of opium was administered. By the 18th a swelling appeared in the right iliac fossa and the diagnosis of perityphlitis was confirmed. Sir Francis Laking said that another surgeon should be called. The King flew into a rage because it was now eight days for the Coronation, which was fixed for the 26th June, 1902, and ordered Sir Francis out of the room. Later the King felt sorry, called Laking back and apologised and agreed that Sir Frederick Treves be called to Windsor. Treves confirmed the diagnosis but advised waiting until the abscess should get walled off. On Saturday the 21st temperature dropped to normal and the swelling receded. On Monday the 23rd the King travelled by rail to London, and London rejoiced that the Coronation would not be put off. But on the 24th he had pain again with fever and vomiting. An operation became inevitable. The decision was taken at 10 a.m. He was operated upon at twelve noon on 25th June, 1902, and a Bulletin to that effect was issued under the signatures of Lister Thomas Smith, Francis Laking, Thomas Barlow. Sir Fredrick Treves cut down on the abscess, put in two drainage tubes and packed the wound with iodoform gauze. It was ten days before the King was out of danger and recovered completely on 9th August.

It is difficult to conjecture what would have been the verdict of the medical profession, had the King died. At this distance of time we cannot judge. We must remember that all concerned were but the children of the time they lived in. Only Murphy and the American surgeons were ahead of their time, and a handful of English surgeons were beginning to wake up. Now things have changed. You just utter the word appendix to some of these wholesale appendix merchants and reflexly their hands itch to take it out. The story goes that one senior surgeon had a reference book with a beautiful index and some useful information added as an appendix. He lent his book to a junior surgeon with itching hands. The book was duly returned but the appendix had been removed.

Surgery broke down one barrier after another. The heart was the last hurdle to be got over, and the credit goes to Louis Rehn.

One young German gardener in Frankfurt was stabbed in the chest on 7th September, 1896. A policeman brought him to the hospital and one Dr. Siegel saw him. He shook his head as the policeman asked him what chance the poor chap had to survive. Had not the great Billroth said, "The surgeon who ever attempts to stitch up a wound in the heart may be certain that he will lose all his colleagues' respect for ever"? Dr. Louis Rehn the surgeon of the hospital was out of town and was not returning for another two days. When Dr. Rehn returned he saw the patient in a very critical state. For two thousand years medical writers had stated that heart wounds are always fatal and that any attempt to touch the heart with an instrument would cause it to stop. He debated the question. Must the man die? Could not all these medical writers have just perpetuated a myth? He recalled having read that the great French army surgeon Larrey had opened the chest in 1810 for a stab wound. He had actually touched the heart but not stitched it. The man died some time later. This proved that mere instrumental or digital contact with the heart had not caused cardiac arrest. In 1872 a London surgeon Callender had removed a needle from the heart. The man had stuck a needle into his coat. He was involved in a fight and a blow on the chest caused the needle to pierce the chest wall and go right down to the heart. He drew it out and the heart kept beating. No one so far had stitched up a heart wound. He, however recalled the experimentally made wounds in the heart were stitched up in case of a rabbit and a dog. Rehn took courage in both hands, opened the chest and found that it contained blood. He opened the pericardium, saw the rent in the middle of the right ventricle and stitched it up. Rehn worked night and day on the case. Because of a pleural effusion, the patient ran a temperature. This had to be evacuated. The patient eventually recovered after three weeks and Rehn reported the first successful suture of the heart to 68th annual meeting of German scientists and physicians at Frankfurt. But one surgeon who exposed the heart in a stab case had remained unsung. He was the Negro surgeon Dr. Daniel Hale Williams of Provident Hospital, Chicago. The colour bar deprived him of any priority. Now as we all know it is possible to do open heart surgery with extracorporeal circulation. We dare not predict what surgery cannot do. All predictions so far have been falsified. You may well ask *Quo vadis* to surgery?

THE ROLE OF SOCIAL MEDICINE IN MEDICAL PRACTICE AND NATIONAL PLANNING OF INDIA

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INTRODUCTION

Medicine is an applied bioscience dealing with men, while medical and health services are essentially social institutions. Hence social medicine should be taught to students of medicine and paramedical sciences. Medical students are taught biochemistry and biophysics but not biosocial sciences which are equally important. As doctors have to deal with human beings they have to study the biological and sociological problems of health and disease. Most of our health problems are sociological rather than biological. For this purpose doctors should be oriented as social scientists with a human outlook and helped by social workers particularly in rural areas. As doctors are more interested in the study of disease, they should think of the patient as well as his disease. The 'human values' of man should not be neglected by doctors.

Doctors are taught more of disease than health in their training. Textbooks of medicine have titles such as the diseases of chest, abdomen, or pelvis, heart, lungs, kidneys, or bones; diseases of women and children; etc. Textbooks do not start with the title such as "Health of . . . ". A minister is called a Minister of Health, and not a Minister of Disease. Medical practitioners must

recognise that they are practitioners of health as well as of disease. Health and disease are but a continuous process, no one is 100 per cent healthy or 100 per cent diseased. When men drift from health to disease, the doctors should cure the disease and restore them to health. No doubt physiologists in preclinical course teach the normal functions of the body but when students cross over to the clinical course, the pathologists overwhelm them from the normal to the abnormal. As pathology forms the background for the three main subjects, medicine, surgery, obstetrics and gynaecology, students emerge out of the college with a 'disease' outlook.

During the last 5 years, we have had several conferences, committees and seminars on undergraduate medical education at all levels; and while we are slow in implementing their recommendations, we are in a great hurry to increase the number of medical colleges for undergraduates without having experienced teachers. We are even increasing and organising study in postgraduate degrees and diplomas, even in preventive and social medicine. The Silver Jubilee Session of the Indian Medical Council (February, 1959) intended to review only postgraduate education, properly confined themselves to undergraduate level in this discipline, which is still far from perfect. They formed a sub-committee to emphasise teaching of 'basic biosocial sciences' in the pre-clinical curriculum.

Can we blame the freshly passed graduates who even in the internee period instead of consolidating their 5-year undergraduate course, think only of postgraduate course, degrees and diplomas? Can we blame students who study for the sake of passing examination and not for acquiring knowledge and fostering a critical study of principles and developing independent lines of thought? Can we blame our 'part-time' teachers who, swamped by their private practice, cannot devote 'whole-time' to teaching and training? Can full-time teachers, after teaching for 20 years on the same subject, confidently say that they need not prepare for even two hours for delivering an one-hour lecture, to keep abreast of current knowledge, to present the subject with a different approach every year. Teachers of preventive and social medicine are among the few full-time teachers in medical colleges, and are treated with suspicion and neglect. How can social medicine be adequately taught or practised and train the large number of field workers required for the National Five-Year Plans? If the Second Five-Year Plan Rural Schemes are analysed objectively, can one doubt their wisdom and recommendations?

Biosocial Approach to Medicine

During the preclinical courses, the medical student must be oriented towards the biosocial approach to medicine, i.e., the behavioural sciences which include sociology, social anthropology, social physiology, social pathology, social and preventive paediatrics and psychology. These subjects can be taught as adjuncts to the various disciplines by the heads of respective departments and/or by departments of preventive and social medicine. Sociological problems must precede biological problems of the disease in the preclinical course. Otherwise the curative aspects in the clinical course will fix themselves like the 'fixed-virus' to the neurons of the medical students' knowledge, and nothing can be done later to prevent this. 'Prevention is not only better but cheaper than cure', particularly in an underdeveloped country like ours. Preventive and social medicine must be taught both in the colleges and in the field, and should be integrated with almost every discipline and department of the college. Part I, i.e., biosocial sciences should be taught at the end of the clinical course, and Part II later, to include others.

The medical colleges have on their staff freshly passed graduates who are called professors. As they pass out taking D.P.H., or B.S.Sc. they qualify themselves for administrative service as Health Officers or by promotion in administrative services as Assistant Director of Public Health or Retired Directors of Public Health (as in this State), but such selections are not made on academic grounds, whereas the Indian Medical Council lays down 10 years' experience for professors in all other departments. It is no wonder that the departments of preventive and social medicine in Indian medical colleges did no more than change their name from hygiene and public health, which taught only environmental sanitation, but nothing of the new course and curricula as envisaged by recent conferences, committees and seminars. To add to these, there is yet no textbook in India with the title 'Preventive and Social Medicine', and there is no universally approved curriculum and syllabus, which changes from one university to another and even from college to college.

Epidemiological Approach to Preventive and Social Medicine

Life is but a continuous and continued alteration and adaptation of the living to the ever-changing environment. Hence health and disease are influenced by three factors: (a) host

(man), (b) agent (bacteria and parasites) as well as (c) environmental factors, including physical, biological, psychological, social and cultural. While the host-agent relationships are sufficient and suitable for the study of curative medicine, by considerations of host-agent balance alone, students of preventive and social medicine must study all the environmental factors, and the interactions between them and with the host and the agent. This is the study of epidemiology, which started from Hippocratic era with study of epidemics alone, and later extended to endemics or chronic diseases, and now applied to the study of every disability and deformity, such as diseases of unknown aetiology like cancer, diabetes and hypertension, and even to suicides, snake-bites and accidents. Epidemiologists and biostatisticians take all the facts and figures of any disease into consideration, analyse them and arrive at constructive data and make recommendations for cure as well as prevention. In the past, while hygiene, sanitation and public health were taught, we were concerned mainly with physical environments alone, i.e., environmental sanitation relating to water supply and disposal of waste products, etc. But when we drifted to preventive medicine we concentrated mainly on acute infectious diseases. Now we recognise that our health status lies more in chronic endemic social diseases, and we shifted to social medicine i.e., the study of total disease, in the total man in the total environment.

One may ask why we use social medicine in addition to preventive medicine? Modern society, particularly after World War II, has so many new complex problems caused by rapid and unplanned urbanisation, high levels of living index with abnormal increase in the three essentials of life such as food, housing and clothing ; and break up of attachments to joint-family system with smaller families. The family doctor who studied the total disease in total environment disappeared with the large family, and is replaced by expert and hurried consultants. This has brought to focus the need for social medicine, replacing preventive medicine and hence the need for study of socio-economic factors in the new cultural background of society. Why we do not yet teach epidemiology in our colleges is because we had no epidemiologists before. It is only a month ago that an epidemiologist was appointed as the Director General of Health Services at New Delhi ; the State Governments will probably take another decade more to do so. An epidemiologist-biostatistician must be found on the staff of these departments like the medicosocial workers and health educationists ; but very few of them are yet staffed on this pattern.

Social Medicine in Medical Practice

Medical practitioners of today are required to shoulder greater responsibility than in the past. As they have to diagnose early cases of chronic diseases and trace them to their environment, they have now to use a variety of methods and laboratory techniques for early diagnosis and treatment. Instead of getting quick and spectacular results with chemotherapeutic drugs and antibiotics they should now become practitioners of health as well as of disease. Most diseases start in their environments and most patients are sent mack from hospitals to the same environment where they get a relapse of the disease. Treatment of the environment is social pathology which is never taught nor even well understood today. We must revert back to the old family doctor who, when called to see a patient, saw the whole family and enquired about all the factors which caused the disease—both biological and socio-economic, in their natural environments of the family or factory. Doctors must afford the time and trouble, and patients or State must afford the money, to arrange a periodical health check-up and get at early diagnosis and treatment which apply as much to curative medicine in all its branches as to preventive medicine. One should find out the causative factors of the disease ; but most diseases are multicausal and multiphased. Has the staff of the public health services in India the time to unravel the multicausal nature of diseases? The acute infectious diseases form only 0·1 per cent of the total mortality and 1 per cent of total morbidity? How many public health officers are research-minded or have research qualifications and equipment to detect the causation of chronic endemic diseases which are mostly social diseases? Medical practitioners alone can fill this gap, while the Government health officers can do no more than what they are doing at present. By re-orienting their outlook and becoming practitioners of health as well as practitioners of disease, the family doctor can make an early diagnosis and offer treatment. This can be accomplished if every district branch of the Indian Medical Association can organise a diagnostic laboratory which all medical practitioners can use on a co-operative basis and get quick reports on specimens sent there. Will the government help the district I.M.A. in this endeavour by subsidising such laboratories?

Social Medicine in National Planning

It is not a mere accident that the first All-India Medical Education Conference and the first and last meeting of the Health Panel of the Planning

Commission which met in New Delhi in September/October, 1955, drew up the Health Plans for the Second Five-Year Plan, as well as recommended the departments of preventive and social medicine in Indian medical colleges. Without this the training programmes for re-orienting medical officers for primary health centres, auxiliary health workers, sanitary inspectors, health visitors and nurses and midwives, the plan cannot work at all. Although the Second Five-Year Plan is soon ending, the Health Panel of the Planning Commission never met again, to assess the results of their recommendations! Although the Third Five-Year Plan is getting ready by now, the lessons learnt from Second Five-Year Plan are not available. Will the Government appoint an assessment committee to see how far departments of preventive and social medicine in about 20 out of the existing 55 and odd medical colleges, functioned since 1955, and how far the trained personnel envisaged for the National Health Plans are adequate in quantity and suitable in quality?

Summary

Doctors and others must consider human beings who were once healthy but now drifted to disease, as both biological and social beings. This can be done best, if they are taught both biological and social factors in health and disease.

Medical practitioners as well as medical and health personnel must study the normal behaviour of man before they impose their prescriptions and skill on him. Patients are to be studied in their natural environment—physical, biological, psychological, social and cultural. This biosocial and epidemiological approach will help the doctors and officers and services to be more efficient and economical.

The transition from hygiene, sanitation and public health, preventive medicine to social medicine, the history of the progress of medicine are the result of the advancement of our knowledge and the needs of the population. During the last century preventive medicine with the prevention of epidemics and acute infectious diseases, could not solve all our health problems. After World War II, social medicine came in as patterns of society changed ; and with it new problems arose, not merely biological but social and socio-economic and cultural.

Prevention is not only better but cheaper than cure, for an underdeveloped country like India. The medical sciences can do no more than give temporary cure without 'curing the environment'. The health services can offer no more than environmental sanitation, in which they did not fully succeed and in rural areas several problems need research in the multi-causation of diseases. Removal of causes is true prevention.

The success of National Health Plans—second five-year Plan and third five-year Plan—depends to a large extent on training of personnel for rural health services in departments of preventive and social medicine. How far these departments have succeeded in the second five-year Plan must be assessed forthwith, before framing plans for the third five-year Plan.*

CASE NOTES

ABDOMINAL PREGNANCY

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Abdominal pregnancy though rare is a wellknown condition. The ratio of abdominal pregnancies to total deliveries in the literature varies from 1 :286 to 1 :15000. The first case seen in the Central Hospital, Dhanbad during the last six years, in about 2500 deliveries, is reported below.

Case Report

Mrs. Turi, aged 30, was admitted to the maternity ward of the Central Hospital, Dhanbad on 11-3-59.

Present history—She was admitted for pain in the abdomen and vaginal bleeding with a history of amenorrhoea for 9 months.

Obstetric history—Full term normal delivery, alive, 10 years ago. No issue after that.

On examination—She was found to be thin and ill-nourished with pulse rate 90 per minute and respiration 20 per minute, temperature 101°F and B.P. 90/60 mm. Hg. In the heart and lungs, no abnormality was detected. Her general condition was very poor.

P.A.—There was enlargement of the abdomen up to xiphisternum ; which was partly cystic and partly solid to the feel, no definite foetal parts being discernible.

The patient admitted to have felt foetal movements which stopped a fortnight before admission. Abdomen was tense and there was slight oedema of the abdominal wall. Presentation of the foetus

* Based on a paper read at the scientific session of the third Andhra Pradesh Medical Conference, Eluru (Andhra Pradesh), February 1960.

could not be made out. The abdominal contour looked normal.

P.V.—Cervix was not effaced ; os was tightly closed ; vertex presenting. No placenta felt. There was not much active bleeding ; small blood clots which were present were removed.

Management and progress—In view of her general low condition she was kept under observation. She was given intravenous infusions, e.g., osmoplasma, glucose-saline solution, vitamin C. Sedatives and antibiotics (penicillin) were also given. Her blood was examined and blood grouping and cross-matching were done. The patient had Hb. 55 per cent (Sahli) ; R.B.C. 2·8 mill/c.mm.

Her condition was gradually improving but the size of the abdomen steadily increased. The temperature became normal after 12 days. On 23-3-59, artificial rupture of the membranes was tried but it could not be done as the os was tightly closed. She was given medical induction of labour on 31-3-59 with no effect. On 1-4-59 induction of labour was again tried with pitocin, and this time she had very slight pains which, however, stopped on 3-4-59. The foetal parts could not be felt and she was x-rayed on 4-4-59.

X-ray showed foetal parts and Spalding's sign was positive.

She was put on general lines of treatment and had all-round improvement. No drastic interference was thought of. On 15-4-59 another internal examination was made which revealed the following : Cervix was small, fibrotic, and pushed high anteriorly, abdomen was tense and increased in size.

Artificial rupture of the membrane was attempted under vision but this could not be done. The patient complained of abdominal discomfort. Abdominal pregnancy was diagnosed and laparotomy was proposed

Operation—On 15-4-59, the operation was performed with arrangement for blood transfusion. Spinal heavy nupercain, 1·6 ml., was given. An injection of pethidine hydrochlor 50 mg. was given during the operation.

The abdomen was opened through right paramedian incision. The anterior parietal peritoneum was found adherent to the amniotic sac (gestation sac). It was separated carefully and incised in the line of the abdominal incision. The sac was markedly thickened, so much so, that at first sight it was thought to be a thinned-out uterine wall. It was opened by longitudinal incision. A large quantity of thickened, chocolate coloured liquor amnii was drained. The gestation sac was found adherent to the peritoneum, omentum and small intestines on all sides. The foetus was lying in the right half of the sac. It was a full-term male foetus. The foetus and the placenta were found macerated and firmly adherent to the gestation sac from within. It was separated from the sac with great difficulty and removed. The whole of the scalp, right ear, lips and chest were adherent to the sac on the right side. After delivery of the foetus there was some room in the abdomen and the amniotic sac could now be pulled out a little. It was gradually and carefully separated from the greater omentum and the small intestines by blunt dissection with sponge towels, and mounted gauze sponges. The uterus was of normal size and the sac arose from its right cornua. The right tube appeared to be stretched over and merged into the sac, the left tube and ovary were normal. The sac, firmly adherent to the fundus of the uterus, was removed along with the placenta. The remnants were removed completely piecemeal. Fundectomy was done as the fundus of the uterus was unhealthy, and sutured. The abdominal cavity was toiletted. The raw areas of the bowels were peritonised. The adhesions were separated gently and then the abdomen was closed in layers in the usual manner after putting in a drainage tube.

The patient was given intravenous infusions of blood 250 ml., periston 540 ml., and glucose-saline 540 ml.—all by the drip method.

Penicillin and streptomycin were given for 5 days. Drainage tube was removed after 48 hours. Temperature came to normal after 4 days. Stitches were removed on 24th April. There was good union. On the whole the patient had an uneventful post-operative period, and was scheduled for discharge on 27th April, 1959. But, she got diarrhoea from food poisoning and was accordingly treated. She was finally discharged from the hospital on 4th May, 1959 in perfect condition.

Discussion

It might fairly be asked "Why was the condition not diagnosed soon after admission." Not being particularly intelligent, the patient failed to give proper history of pain during early pregnancy. Her abdominal contour was normal. The cervix was not much displaced anteriorly in the beginning, although it was rather small and tightly closed. It was thought to be a probable sequela of cervical erosion. Her general condition was low and she was running a fairly high temperature. Hence more attention was paid to the first condition.

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Most cases of abdominal pregnancy do not present a classical picture. Some of the signs and symptoms thought to be helpful in diagnosis are :

1. An abdominal crisis early in pregnancy.
2. Palpation of the superficial foetal small parts.
3. Unusually loud foetal heart tones.
4. Inability to palpate the round ligaments and absence of Braxton Hicks contractions.
5. False labour.
6. Persistent abdominal pain or tenderness.
7. Definite displacement of the cervix.
8. High position or transverse lie of the foetus.
9. Signs of foetal death.
10. Identification of an extrauterine mass.

In the present case (i) persistent abdominal pain and discomfort, (ii) a small, fibrotic cervix which was later on displaced anteriorly, (iii) intrauterine death of the foetus, and (iv) unexplained vaginal bleeding helped in establishing the diagnosis. However, the correct diagnosis of an abdominal pregnancy rests chiefly on a high index of suspicion on the part of the clinician.

There is some disagreement in the literature concerning the management of the patient with abdominal pregnancy. If the foetal death has occurred, some authors prefer to wait from seven days to eight weeks until the placental circulation has considerably decreased, minimising the danger of haemorrhage at the time of operation. Others feel that such delay results only in an increased susceptibility to infection with a resultant increase in the maternal morbidity and mortality. The management of placenta should be individualised. The decision whether to remove the placenta or not has to be influenced by the location and condition of the placenta, condition of the patient, and availability of whole blood. In the present case, the foetus and placenta were macerated. Therefore, the chances of haemorrhage were less, as the circulation had stopped long ago. The placenta could be removed completely. As there was not much of haemorrhage, and the patient received blood, there was speedy and uneventful recovery.

Summary

A case is described of secondary abdominal pregnancy proceeding to term in a 2-gravida aged 30. The foetus was found macerated. The placenta and the sac could be removed completely at the time of operation. The patient had an uneventful recovery.

Acknowledgment

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HYPOTENSION DUE TO MEPROBAMATE SENSITIVITY

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When the tranquillising drug meprobamate (2-methyl, 2-N-propyl-1,3-propanediol dicarbamate; equanil; miltown) was introduced as a therapeutic agent it was thought to be relatively innocuous, and it was suggested that large amounts of the drug could be taken without any serious ill-effect. Borrus (1955) mentioned many side-effects of this drug in therapeutic doses. In his series some patients became very drowsy while in others it had no effect. Since then about 18 cases of coma due to attempted suicide with meprobamate have been reported (Charet *et al*, 1956; Heberden and Cooper, 1957; Shane and Hirsch, 1956; Collins-Dineen, 1956; Hiestand, 1956; Woodward, 1957; Bedson, 1959; and Stevens, 1960). All these cases showed only a mild fall in blood pressure, except in cases described by Woodward, Bedson and Stevens. In Woodward's patient, the systolic pressure fell to 40 mm. Hg and the patient remained unconscious for 44 hours after taking 96 tablets (38·4 g.). Bedson's patient took 20 tablets and remained in coma for 27 hours, his systolic blood pressure reading 80 mm. Hg. Stevens' patient showed a fall in blood pressure out of proportion to unconsciousness. All the patients described were under the effect of heavy doses of the drug and admitted in various hospitals in unconscious state. The following case is very interesting as he developed profound hypotension with half a tablet (200 mg.) of 'miltown' given as tranquilliser during his stay in the hospital for treatment of his other illness. Although attention has been drawn to the dangers of meprobamate (Editorial, *Brit. M.J.*, 1958), there is no record in the literature of hypotension developing with such a small dose of meprobamate.

Case Report

M. L., aged 82, was admitted in U. H. M. Hospital, Kanpur, in June 1957. He was suffering from periodic distention of the abdomen with difficulty in passing flatus, and constipation. He complained of missing of heart beats coming in attacks.

Past illness—The patient suffered from ulcer and hyperacidity for many years. His latest barium meal examination showed cowhorn deformity of the stomach but there was no obstructive lesion. He had been suffering from constipation for the 45 years and has been taking enema regularly, since that period. In 1955 he once passed one big mucous, rope-like long piece and had developed obstruction at that time which was relieved.

Present illness—Patient was tall and thin built. The pulse rate was between 80 and 96 per minute.

Occasionally he developed missing of beats. When he felt a reeling sensation and syncope. Blood pressure varied between 118/70 and 128/80 mm./Hg. There was no abnormality in the circulatory and respiratory systems. Abdomen showed slight tenderness in the right hypochondrium. Occasionally his abdomen got distended, but no particular loop of intestine was felt and there was no marked visible peristalsis.

He had moderate anaemia—the count being R.B.C. 3·9 million/c.mm ; Hb. 85 per cent. E.C.G. showed nodal rhythm. There was no myocardial defect. X-ray of the chest was normal. He occasionally felt much excited and worried. It was decided to try 'miltown' as tranquilliser. On 25th June 1957 he was given half a tablet of 'miltown' at 8 P.M. as a trial.

On the same night at about 1 A.M., I was called to see him, as the nurse thought him to be in an unconscious state. She had come to give him milk feed, and could not arouse him. I found him in stuporous condition, but he responded to loud interrogations and painful stimuli. His respiration was rapid, 35 per minute, stertorous. Pulse was 54 per minute, with irregular missing of beats. B.P. was 70/50 mm. Hg. Pupil on the right side was moderately dilated and reacting to light ; on the left side it could not be judged due to corneal opacity. Corneal and other reflexes were present. Glucose solution 25 per cent, 100 ml., was administered intravenously. Ephedrine ½ gr., and nikethamide 2 ml., were injected stat and advised to be repeated every 4 hours. His blood pressure did not improve and at 5 A.M., it fell down to 60/35 mm. Hg. The pulse was felt with difficulty ; the heart rate was 60 per minute but still irregular. A 5 per cent glucose solution in normal saline drip with l-noradrenaline, 4 ml. in a pint (570 ml.) was started. The B.P. responded, the systolic pressure rose to 102 mm. Hg. and the pulse rate improved (70 per minute). She was given two pints of 5 per cent glucose solution in normal saline. In the evening the blood pressure again came down to 90/50, and 25 per cent glucose solution, 100 ml., was repeated and ephedrine, ½ gr. with 2 ml. of nikethamide, was repeated and continued at 6-hourly interval. It took nearly 55 hours for his blood pressure to come to the usual normal levels. He developed oliguria during this episode which continued for 38 hours.

Discussion

This case was unusual as the patient developed marked and prolonged hypotension, oliguria, bradycardia, and stupor after a small dose (200 mg.) of meprobamate. His general condition gave indication of the falling of blood pressure. All cases so far described in the literature showed hypotension only with heavy doses of this drug, taken mostly for suicidal purpose.

The second point of interest was bradycardia with intermittent pulse. This has not been a marked feature in cases previously described.

Other interesting features were that though the patient showed good response to l-noradrenaline drip in tiding over the crisis, it took nearly 55 hours before his blood pressure level returned to normal. The period of oliguria was unusually prolonged in this patient. This was probably due to prolonged period of hypotension.

Meprobamate is not a harmless drug, It has been shown to cause habit and produce a typical withdrawal syndrome of insomnia, vomiting, tremors or muscular twitchings, ataxia and anxiety. Acute meprobamate poisoning producing stupor, coma and hypotension have been observed in cases of overdosage of the drug for suicidal purposes, Idiosyncrasy reactions like urticaria, vomiting, fever, oedema, arthralgia and bronchospasm have been described in some cases (incidence being 0·5 per cent). Profound and prolonged hypotension, oliguria, and bradycardia with intermittency of the pulse has not previously been reported to be produced by meprobamate in therapeutic doses. It is probably a sensitivity reaction to the drug demonstrated by this patient. This is the only case of these peculiar sensitivity reactions to meprobamate that the author came across.

Summary

A case developing prolonged hypotension, bradycardia, intermittency of pulse, oliguria and stupor after a single dose of one half tablet of miltown (200 mg.) is described. This is probably due to sensitivity of this drug, not previously described.

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MENTAL MORBIDITY MISUNDERSTOOD

When is the psychiatrist consulted about a patient suffering from mental disease? Certainly when the patient shows some episodic acute symptoms causing considerable disturbance at home. The onset of the illness is then ascribed to some precipitating factor such as failure in an examination, the unsympathetic attitude of relatives, the sarcastic remarks of co-workers and colleagues in the office and perhaps the administration of some poison mixed up with foodstuff or due to some malign influence of some supernatural power. A host of organic explanations is also offered for the condition because no one likes to be considered mentally ill. This may range from head injury, an attack of typhoid fever in childhood, dog bite years ago, to an after-effect of excessive masturbation. The *mental doctor* is not called in first, because the very term 'mental' carries with it a stigma and bears a sinister connotation. These have a deterrent effect on the visit by a psychiatrist. A hush-hush policy is followed and the relatives are sceptical about seeing a mental specialist until the patient himself broadcasts the symptoms of mental disease at the top of his voice and makes a public demonstration of anger, hostility and aggression to the discomfiture of those who wanted to keep it a secret.

At the very first visit immediately after the preliminary examination is over the guardian invariably asks a series of questions—"Doctor, what do you think is the cause of the disease? It is certainly not due to heredity because nobody in our family had ever suffered from mental disease of any kind whatever." And information supplied regarding the inheritance of mental disease must be accepted with due care and caution because few people are aware of the nature of the mental make-up of their forebears. Besides, our knowledge on the subject of inheritance of mental disease has not got a firm footing as yet. However, a predisposition to mental disease may be transmitted from one generation to another as in mental deficiency. The guardian goes on, "Our patient is not suffering from raving madness. You will not be able to detect anything wrong with him when you talk to him. He will answer all your questions to the point. Oh, he was such a nice boy before, very obedient and he never mixed with bad boys. He has a perfect character." These assertions have to be analysed one by one. The guardian is not aware of the fact that the onset of the mental disease is frequently slow and insidious. So he did not perhaps observe some subtle changes in the patient's behaviour long before the onset of the acute episode. The diagnosis of mental disease does not depend primarily on asking questions and hearing the patient's answers. Mental disease is easier recognised by observing the patient's attitude and behaviour and by noticing the presence of emotional disturbances and storms. What he says is not so important as how he says it.

As regards obedience and 'nice' character it will not be wrong to surmise that the patient was so much repressed and inhibited that he could not express his desires and fears freely owing to the dominance of the guardians. Moreover, a person who is shy, sensitive, seclusive and an introvert is not likely to be able to mix with people with courage and confidence.

The relatives often enquire whether the psychiatrist ever came across a case of this kind before. Obviously they hint at the peculiar and unique character of the malady. They have to be reassured that this kind of disease far from being rare has become exceedingly common nowadays and that the incidence has been mounting in recent times by leaps and bounds. Then they will ask the psychiatrist about the diagnosis of the case. There is a lurking doubt in their mind about the

diagnosis of mental disease which they hate and dislike to the core of their heart and so they want to get the matter corroborated by a score of doctors. There are people who approach doctors by the dozen, ask their opinion about the diagnosis of the case but they are determined not to follow the instruction of anybody as far as treatment is concerned. Even when the diagnosis is beyond doubt they would prefer to waste time, energy and money on carrying out such ancillary methods of examination and investigations as taking x-ray picture of the skull, examination of the C.S.F. and E.C.G. which only yield negative information.

As regards treatment of mental disease, guardians of mental patients are very often misinformed and ill-advised and are malhandled by interested persons and quacks as well as by unscientific organisations in various ways. There are occasions when institutional treatment becomes absolutely necessary, e.g., when the patient has suicidal ideas or a self-destructive tendency, if he tries to escape from home every now and then, in case of refusal of food and medicine by mouth and when he is aggressive and otherwise unmanageable at home. The question that crops up in such cases is the chance of the patients becoming worse by coming in contact with other mental patients in the institution. Such fears are groundless as most patients improve shortly after admission to hospital principally due to their removal from an uncongenial home atmosphere and to nothing else. The main difficulty lies elsewhere, viz., in the financial position of the patient ; institutional treatment is invariably expensive in our country. It is for this very reason that some patients are taken away from the hospital quite prematurely even before there is partial improvement. Then comes the preoccupation of friends and relations with some specific forms of treatment such as electro-shock therapy, insulin-coma therapy, psychoanalysis and so on. They might even suggest that such and such treatment would do the patient good, instead of giving the psychiatrist a free hand to carry out treatment. They will not rest satisfied even when they see appreciable progress in their patient unless and until the specific form of treatment is undertaken. Sometimes the question of prestige is involved, implying that there will be loss of prestige if the patient is not cured. There was a time when people were mightily afraid of E.C.T. Now the pendulum has swung back to the opposite direction and guardians insist on this form of treatment without considering the indications for it. Parties are known who are obsessed with the idea of psychoanalysis even when the patient is grossly psychotic and stone deaf. Hospitalisation is necessary for enabling the patient to get over safely the acute phase of psychosis.

But the matter does not end there as the patients' friends and relatives are led to believe. They are unaware of the necessity of following up the case. The question of relapse looms large in every case of mental illness if no effort is made to reconstruct the fundamental personality structure by suitable psychotherapy in the post-hospitalisation period. Psychotherapy from this point of view is in the art of influencing personality and character formation. The family to which the patient returns may precipitate the disease again if the relatives lack understanding during the convalescent period.

Hence the members of the family must be re-educated in the management of the patient by social service workers. This measure facilitates the rehabilitation of the patient to a considerable extent. Music, dance, drawings, art work, books and libraries all foster emotional and intellectual insight. These have an important place in psychiatry as they bring to the fore the dynamism and motivation of the patient. This is how emotional re-education, so essential for preventing relapse in mental disease, is promoted. The uncovering of latent qualities in patients enables them to attain social recovery and final rehabilitation. The application of these principles of mental hygiene is a definite forward step for the creation of a healthy mental life and a cheerful outlook on life.

GENERAL PRACTICE DIGEST

MANAGEMENT OF MENOPAUSAL SYMPTOMS

The climacteric or menopausal symptoms result from the cessation of the endocrine function of the ovary. The first effect of waning of ovarian activity is amenorrhoea. The first symptom of the climacteric is, therefore, the menopause. The general manifestations of the climacteric never appear before it. During this period psychological changes appear in the subjects. Many women fear that the change of life is an end of life, especially of sexual life. They fear loss of looks and figure, and of their husband's affection. They fear cancer, insanity, and other serious illnesses. Hot flushes are more troublesome when the patient is anxious, tense, or otherwise emotionally disturbed.

The most common symptoms of the climacteric are hot flushes, night sweats, cold shivers, nervousness, insomnia, depression, irritability, fatigue, and palpitation. Irregular or heavy uterine bleeding is never a manifestation of the menopause. Such abnormal bleeding requires investigation, including curettage and cervical biopsy, to find its cause before treatment is instituted.

General Treatment

Most patients should be told that the disturbances are physiological, temporary and of less account and that sexual capacity need not be impaired, and that a full and active life is not at an end.

Reassurance is necessary for alleviation of climacteric symptoms. Only a small minority (approximately 5 per cent) of patients will need medical treatment. Obesity can be controlled by a strict reduction of the calorific intake. Dieting is good for morale. The number and intensity of hot flushes can be reduced by avoiding hot baths, hot atmospheres, alcohol, coffee, and highly seasoned foods.

Medical Treatment

I. SEDATIVES AND TRANQUILLISERS :

These are given to control irritability and emotional upsets. They help in quieting vasomotor instability and in reducing the number of hot flushes.

Phenobarbitone, gr. ½ (32 mg.), once or twice daily, is the best sedative drug. It may be combined with ascorbic acid and vitamin B complex. Of tranquillisers, promethazine, 10 to 25 mg. each night, or meprobamate, 400 to 800 mg. at night is the best drug for climacteric symptoms.

II. STIMULANTS :

When lethargy and nervous depression are present, sedatives should be avoided, and amphetamine or dextroamphetamine sulphate, 5 to 10 mg., twice daily, recommended.

III. SEX HORMONES :

The object of giving oestrogens should be to give only enough to improve symptoms and to make them tolerable, not to eliminate them. The required dose should be determined by trial—the index being the number of hot flushes, which should be reduced to an average of 4 in 24 hours. When this has been achieved, the dose is maintained at that level for 2 weeks only. Thereafter it is gradually reduced during the next 2 to 3 months. At the end of this period, oestrogen therapy should be discontinued completely and should not be reinstated even though symptoms recur.

Treatment can also be given intermittently, with a break of seven days every three weeks—this will avoid endometrial hyperplasia.

(i) *Oral oestrogen preparation*—The most commonly used preparations are stilboestrol and ethinyl oestradiol. They are cheap, and highly potent. Start with a test dose of stilboestrol, 0·1 mg., two or three times daily. It is rarely necessary to give more than 0·5 mg. in 24 hours and a daily dose of 1 mg. should never be exceeded.

Ethinyl oestradiol is approximately 20 times more powerful than stilboestrol, and not more than 0·05 mg. daily should be given. It is better to give 0·01 mg. once, twice or thrice daily.

Synthetic oestrogens sometimes cause nausea and vomiting; in such cases natural oestrogen should be used. The latter is less potent and very expensive. The most active oral preparation is one of the conjugated oestrogens, obtained from mare's urine. The initial dose should not exceed 1 mg. daily—best divided into two or three doses equally spaced.

For depot treatment chlorotrianisene (a synthetic oestrogen) is given as one capsule of 12 mg., twice daily, for 30 days. The course must be completed (30 days), and then weaned as symptoms are relieved. This is a very expensive preparation. Because of the fear of inducing uterine haemorrhage, chlorotrianisene should be given only to women without a uterus.

Methallenoestril is relatively free from undesirable side-effects. A suitable initial dose is one tablet of 3 mg. once or twice daily. This preparation also is expensive.

(ii) *Oestrogen preparation for intramuscular injection*—For psychological reasons it is generally unnecessary and unwise to prescribe injections in the treatment of climacteric symptoms.

(iii) *Implants*—The subcutaneous or subfascial implantation of tablets of 50 to 100 mg. oestradiol is useful in relatively young women whose uterus or ovaries have to be removed. It provides a depot from which absorption takes place in a gradually decreasing amount over a period of 2 to 6 months.

This treatment should never be applied to a woman with a uterus.

(iv) *Androgens*—These drugs inhibit the gonadotrophic function of the anterior pituitary. They do not stimulate the female secondary sex organs and they cannot cause uterine bleeding. They give a sense of well-being, eliminate depression and increase libido. But they are expensive preparations and are likely to cause hirsuties, acne, voice changes, and other signs of virilism. Androgens should be used in which even small doses of estrogen cause uterine haemorrhage or in which there is previous history of endometriosis or genital cancer.

CURRENT MEDICAL LITERATURE

Antihypertensive Action of Chlorothiazide

JOY, J. T. AND BEISEL, W. R. (*Am. J. M. Sc.*, **239** : 601, 1960) from the Department of Metabolism, Division of Medicine, Walter Reed Army Institute of Research, Washington 12 D.C. write :

Chlorothiazide is a unique diuretic in that it causes a marked loss of body potassium and reduces an elevated blood pressure. It was postulated that the ability of the drug to lower blood pressure might be related to its kaliuretic effect.

A previously untreated 51-year-old patient with uncomplicated "essential hypertension" was studied by means of the metabolic balance technique, and treated with chlorothiazide. His blood pressure was reduced by the drug and he had a concomitant loss of 110 mEq. of body potassium. Repletion of potassium losses did not affect the reduced blood pressure.

Measurements of renal function and renal hemodynamics revealed a fall of the glomerular filtration rate while the drug was being administered. This returned toward its control value when the patient was repleted with potassium. Renal plasma flow and tubular excretory mass remained normal throughout. The renal afferent and efferent arteriolar resistances decreased while chlorothiazide was being administered, but did not increase again when potassium was repleted. The relationship of the decreased renal arteriolar resistance to the primary mechanism of action of chlorothiazide cannot be proven, but is not a function of a decrease in body potassium.

Chloroquine Phosphate in Severe Infectious Mononucleosis

GOTHBERG, L. A. (*J.A.M.A.*, **173** : 53, 1960) of Spokane, Washington writes :

Four patients with infectious mononucleosis, whose cases are described, showed a dramatic, and possibly specific, clinical response to treatment with chloroquine (Aralen) phosphate. Although the possible modes of action of chloroquine phosphate, as outlined here, are not entirely understood, further trial of the drug is warranted because of (1) the gratifying symptomatic relief patients obtained from its use and (2) the light that may be shed on the mechanism of action of the 4-aminoquinolines.

Megaloblastic Anaemia Associated with Anticonvulsant Drugs

FLEXNER, J. M. AND HARTMANN, R. C. (*Am. J. Med.*, **28** : 386, 1960) from the Hematology Service, Department of Medicine, Vanderbilt University School of Medicine and Hospital, Nashville, Tennessee, from two cases of megaloblastic anaemia associated with the use of dilantin and barbiturates, observe :

Detailed nutritional studies indicated poor dietary habits and in particular low intakes of folic acid, vitamins C and B_{12}, and carotene. Urinary formiminoglutamic acid excretion provided further evidence of a folic acid deficiency state in the patient (Case 1) studied. This patient experienced complete clinical and hematological remission with the ingestion of 500 μg. of folic acid daily. Further studies demonstrated no evidence of a malabsorption defect or Addisonian pernicious anaemia.

To date no inhibition of folic acid has been demonstrated when anti-convulsant drugs were added to certain microbiological systems. However, the clinical experience obtained with remissions, following withdrawal of anticonvulsant drugs and relapses upon re-institution of these drugs, leaves little doubt that these agents are concerned with the development of the megaloblastic anaemia. The reported occurrence of macrocytosis without anaemia in a large proportion of epileptic patients receiving these anti-convulsant drugs further supports this conclusion.

A frequent conditioning feature for the development of anticonvulsant megaloblastic anaemia may be the presence of occult nutritional deficiency secondary to an inadequate diet. Some folic acid deficiency state seems paramount. Vitamin C deficiency may play a role in some cases since the dietary sources of ascorbic and folic acids are virtually identical. Mild to moderate vitamin B_{12} deficiency, when present, may augment the megaloblastic disorder but does not appear to be necessary or even frequent. The deficiency in carotene in our patients probably reflects the dietary inadequacy without influencing the megaloblastic picture.

Whether nutritional deficiency is actually a prerequisite for the development of the megaloblastic anaemia remains unsettled. However, the demonstration of some dietary deficiency in many of the well investigated cases and the rarity of frank megaloblastic anaemia among the multitude of epileptic patients receiving these anticonvulsant drugs, suggest that malnutrition frequently plays a role.

Transient Myopia After Acetazolamide

MUIRHEAD, J. F. AND SCHEIE, H. G. (*A.M.A. Arch. Ophth.*, **63** : 375, 1960) from the Department of Ophthalmology, Hospital of the University of Pennsylvania, Philadelphia, and the Children's Hospital of Philadelphia, report :

A case of transient myopia with transient macular oedema following acetazolamide ingestion is reported. Alterations in refractive index, contour, or position of various ocular structures are considered as possible pathophysiological mechanisms. Narrowing of the contour of the anterior chamber angles suggests forward displacement of the iris, which may be due to either increased volume of the ocular contents behind the iris (including the lens) or diminished volume of the anterior chamber. There is insufficient evidence to decide which of the mechanisms is primary.

Acute Renal Insufficiency Following Aortic Surgery

DOOLAN, P. D., WIGGINS, R. A., THIEL, G. B., LEE, K. J. AND MARTINEZ, E. (*Am. J. Med.*, **28** : 895, 1960) from the Clinical Investigation Centre, U.S. Naval Hospital, Oakland, California; from an analysis of the study on eleven patients having acute renal insufficiency following aortic resectional surgery observe :

All the patients were men; eight had aneurysms; three had occlusive vascular disease; and in each in-

stance the lesion was distal to the renal artery. In five of the eight patients the aneurysms had ruptured, emphasising the association of ruptured aneurysm and subsequent renal failure. Six of the patients survived, which is most encouraging in view of the coexistence of significant cardiovascular disease in ten of the eleven patients. A cerebrovascular accident, bronchopneumonia, rupture of the aorta and in two instances refractory hypotension were the principal causes of death in the five fatalities in which uraemia was considered of secondary importance. Extensive renal infarction occurred in three of the patients. Nine of the eleven patients had sustained periods of significant hypotension and the renal failure may have had its origin in this circulatory stress.

The current state of knowledge concerning the pathogenesis of acute renal insufficiency under these circumstances is reviewed and the opinion expressed that efforts directed at the relief of renal vasoconstriction are rational and should be pursued. The recommendation to infiltrate the renal pedicle during aortic surgery, especially in patients in whom rupture of an aneurysm has occurred, is supported.

In three of the eleven patients gangrene of the lower extremity developed, a complication of particular significance in the oliguric subject. In these three patients potassium intoxication developed, and the rate of clinical deterioration seemed out of proportion to the rate of progression of the uraemia. Amputation was performed in all three patients. In the difficult decision regarding the choice of treatment in these or other patients manifesting less obvious signs of vascular insufficiency, assistance has been sought in measurements of the PO_4 : blood urea nitrogen ratio and the plasma-creatine concentration. Both of these values are elevated in the presence of devitaliser muscle but with further studies high values were also found in other patients with uraemia who were free of this complication. The plasma creatine concentration may exceed the creatinine concentration in uraemia. The choice as to conservative management, debridement or amputation in patients with devitalised muscle cannot be made on a basis of biochemical data alone and for the present must remain a matter of judgment.

Malignant Disease in the Aged

ELIAS, R. AND LESNICK, G. J. (*Am. J. M. Sc.*, **239**: 554, 1960) from the Central House Unit of the Home for Aged and Infirm Hebrews of New York, New York, observe:

The incidence and results of management of malignant disease in a home for the aged has been reviewed. Sixteen per cent of patients dying at the Central House Unit of The Home for Aged and Infirm Hebrews of New York had evidence of malignant disease prior to death or at postmortem examination. Three-fourths of them died of the malignancy. Tumours of the gastrointestinal tract comprised 49·6 per cent; the genitourinary tract, 16 per cent; the lungs, 11·9 per cent and the breast, 10·9 per cent. Diagnosis was established prior to death in two-thirds of the patients. In a little over one-third of the patients, diagnosis was established early enough to permit treatment with some hope of success. Treatment gave significant palliation in two-thirds of those treated and resulted in cure of the disease in one-fourth of those treated.

This study indicates the need for simple screening tests, such as blood and stool examination, and frequent weight measurements in older individuals. The excellent response to therapy in many of our cases emphasises the value of early aggressive therapy even at advanced ages.

Acute Gastric Dilatation

LEIGH, T. F. (*J.A.M.A.*, **172**: 1376, 1960) from the Department of Radiology, Emory University School of Medicine, writes:

Acute gastric dilatation is a condition characterised by clinical findings of vomiting, abdominal distention, and collapse. The gravity of this condition in most instances makes mandatory its prompt recognition and treatment. The diagnosis can be quickly and easily established by proper radiographic examination of the abdomen. The necessary examination can be carried out at the bedside as well as in the radiology department. The radiographic examination further aids in the differentiation of this condition from other abdominal conditions which show similar clinical findings.

Cholecystography with Bunamiodyl

MESZAROS, W. T. AND RICH, F. M. (*J.A.M.A.*, **172**: 1884, 1960) from the Department of Radiology, Cook County Hospital, Chicago, write:

A new orally given contrast medium, bunamiodyl, was used in 500 consecutive patients referred for routine cholecystography. The order and severity of side-effects was less than that observed by ourselves and, apparently, by others when compared with several other commonly used contrast agents. The quality and intensity of contrast values with use of a standard six-capsule dose (4·5 g.), regardless of patient weight, were satisfactory.

Unabsorbed colonic residual medium, often confusing when other mediums are employed, is minimal with bunamiodyl. If it does occur, it is usually diffuse in character and does not interfere with film interpretation.

On the basis of this evaluation, bunamiodyl is believed to be a safe and efficient orally given cholecystographic contrast medium. Fewer side-effects and some technical improvement over presently used agents were observed.

Experimental Vaccination Against Measles

SCHWARZ, ANTON J. F., BOVER, P. A., ZIEBEL, L. W. AND YORK, C. J. (*J.A.M.A.*, **173**: 861, 1960) from the Research Division, Pitman-Moore Company write:

The antigenicity and safety of modified live virus measles vaccine was studied in monkeys and in two children. None of the monkeys inoculated orally, intramuscularly, or intercerebrally showed signs of disease, and all produced antibodies. Two children inoculated with the vaccine had a good immunologic response but developed a mild illness with fever, sore throat, and some leukopenia. Distemper virus, believed to be anti-

genically related to measles virus, was also tested by intramuscular and intercerebral inoculation into susceptible monkey. No signs of illness were observed, and all monkeys tested developed antibodies against distemper. None produced measles-neutralising antibodies even after two injections of distemper virus. It would appear that the strain of distemper virus used in these experiments lacked the antigenic component which would cause significant measles antibody production.

* * * *

HOEKENGA, M. T., SCHWARZ, ANTON J. F., PALMA, H. C. and BOYER, P. A. (*J.A.M.A.*, **173** : 868, 1960) from the Chiriqui Land Company (United Fruit Company) and the Research Division, Pitman-Moore Company, write :

During a measles epidemic in Panama, 453 persons with no previous history of measles were inoculated with a modified live-virus measles vaccine, and 356 others served as controls. About 70 to 80 per cent of children under 5 years of age had some degree of fever, and about 50 per cent of these developed a slight macular rash. During the following four and one-half months, three of the vaccinated group developed measles, compared with 32 among the controls. In addition, 388 persons were vaccinated with canine distemper vaccine, with 414 controls. None showed reactions to the vaccine. In the following three to four months, 14 cases of measles occurred in the vaccinated group and 25 among the controls. It is concluded that the measles vaccine was effective but caused some reaction, while the ability of this strain of distemper virus to protect against measles appears to be low.

Mouth-To-Mask Resuscitation

TOMASHEFSKI, J. F. AND OLIVER, T. K., JR. (*J.A.M.A.*, **172** : 1888, 1960) from the Cardiopulmonary Laboratory, Ohio Tuberculosis Hospital, and the Pulmonary Laboratory, Children's Hospital, Columbus, Ohio, write :

An instrument which circumvents objectionable aspects of mouth-to-mouth contact when this method of resuscitation is used has been devised. A simple nose and mouth mask, a flutter valve, and an outflow valve prevent the flow of expired air from the injured person to the operator. An inflow valve on the operator's side of the flutter valve provides fresh air. The length of the tube allows the operator to first rebreathe 300 c.c. of his previously exhaled air and prevents him from hyperventilating. A relief valve opens when ventilation pressures exceed 24 mm. Hg. The mechanical and physiological efficacy was checked by laboratory testing of the exchanged air and of blood samples drawn from five subjects during actual resuscitative conditions. The mouth-to-mask method of artificial respiration proves to be safe, simple, and efficient.

Salt Depletion in Infants with Cystic Diseases of the Pancreas

DI SANT'AGNESE, P. A. (*J.A.M.A.*, **172** : 2014, 1960) from the Department of Pediatrics, Columbia University, and the Babies' Hospital, New York, gives in the following lines the summary of his observations :

Patients with cystic fibrosis are known to be unusually susceptible to hot weather, because of massive salt depletion through the sweat glands. It is not generally appreciated that under certain circumstances infants with cystic fibrosis are prone to salt depletion and dehydration with serious consequences even in cold weather.

Five such cases are reported, in four of which the diagnosis of fibrocystic disease was not known or even suspected prior to admission.

On the bases of observation and of studies performed, it was concluded that in these patients renal and adrenal function were normal. The mechanism of salt depletion was thought to be repeated small excessive losses through the sweat and possibly in gastric secretions as well, coupled with inadequate oral intake, gradually leading to negative salt balance over a period of time. Under these circumstances patients with cystic fibrosis are especially apt to develop sodium chloride deficiency of severe degree because of the abnormally high electrolyte content of the secretions of their exocrine glands.

It is important to keep cystic fibrosis in mind in the differential diagnosis of hypoelectrolytaemia and dehydration in infancy, especially when the clinical picture is not adequately explained by the amount of vomiting or diarrhoea. Cystic fibrosis is at least as common as adrenal insufficiency as a cause of this symptom complex in this age group.

From the practical, prophylactic standpoint, it is suggested that as soon as the diagnosis of cystic fibrosis is suspected in an infant, supplemental amount of sodium chloride be added to the diet, even in cold weather. The additional amount administered orally should at least equal the infant's daily requirements and can be given in the formula. Special attention should be paid in such patients to the electrolyte balance when even minor degrees of vomiting develop.

Elevated concentrations of sodium and chloride were found in the breast milk of the mother of one of these patients, despite the fact that she had been taking a low-salt diet for many years.

Management of the Child with Thyroidal Carcinoma

HAYLES, A. B., KENNEDY, R. L. J., BRAHRS, O. H. AND WOOLNER, L. B. (*J.A.M.A.*, **173** : 21, 1960) from the Mayo Clinic and the Mayo Foundation, write that carcinoma of the thyroid in children is a relatively rare condition which has been recognised with increased frequency during recent years. It occurs more frequently among girls than boys, the ratio being 2 to 1 in a series of 59 cases. The diagnosis is established by biopsy, but the disease should be suspected in any child with nodular goiter or persistent cervical adenopathy. Carcinoma of the thyroid in children almost always grows slowly, although metastasis occurs early and may be extensive at the time the disease is recognised. The method of treatment depends on the type and extent of the carcinoma. Whenever possible, all carcinomas should be surgically excised. When complete surgical excision is impossible, radioiodine (I^{131}) should be given, provided the tumour will accumulate iodine. Tumours that will not accumulate iodine should be treated with roentgen rays or radium.

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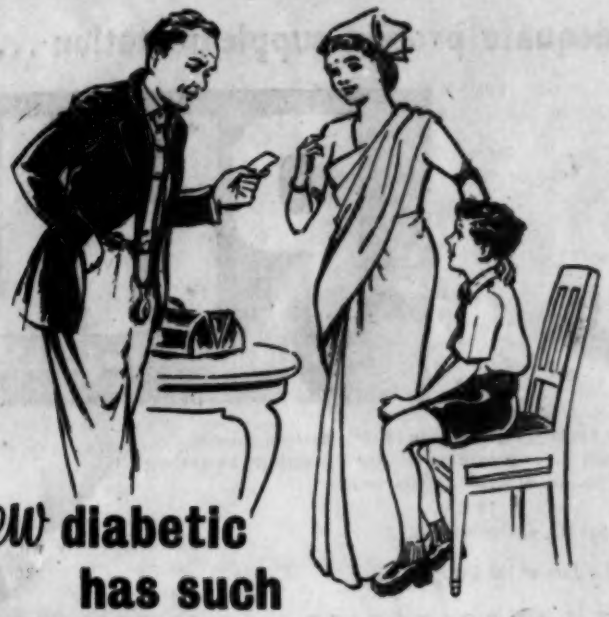

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CURRENT TOPIC

NUTRITION, SANITATION "MUST HAVE TOP PRIORITY IN S.E. ASIA"

Despite a steady reduction of mortality in the S.E. Asia Region the burden of sickness remains very heavy indeed. It is, in fact—says Dr. C. Mani, Director of WHO's South East Asia Regional Office—"the most distressing public health problem of this Region".

Dr. Mani, in his annual review of health progress, presented at the 13th session of the WHO Regional Committee blames "Poverty's twins—malnutrition and insanitation" for this "deplorable state of public health."

He continues: "While governments in the Region are making strong efforts to reduce poverty, through various degrees of industrialisation, the sinister role of malnutrition and insanitation on the health and working capacity of the people is still not properly appreciated. *"The loss of working time is really staggering and until government planning bodies realise this and are willing to give top priority to the improvement of these two vital conditions, this colossal drain on national economics will continue and grow steadily through the coming years of rapid industrial development. . . . ."*

With regard to *malnutrition*, Dr. Mani says that the current tendency is to expect rather much from the education of the public and so responsibility is pushed on to Health Departments without realising that nutrition education—which, after all, is health education—is a slow process extending over many years.

What is needed *urgently*, says Dr. Mani, is "the formulation of dynamic national food production policies which will increase the total production of not only food grains such as rice and wheat, *but also of such nutrients as milk, eggs, fish and vegetables, to a point where the poor can afford to buy them.*

"There must be some reasonable ratio between the quality and quantity of food available for a people. It is not sufficient just to make available enough rice to fill the stomach."

• • • •

As a result of lack of *sanitation* there are the salmonella infections, including typhoid fevers, the dysenteries and intestinal worms (especially hookworm and roundworm) which place millions on the sick list every year.

Ceylon (a small country but one having a very good network of health services) still has twenty thousand cases of typhoid a year. The "same sad picture" is seen on a much larger scale in India and Indonesia. One investigator has estimated that in Thailand a quarter of the population have hookworm as well as roundworm infection. In the north east provinces of the country alone there are about three million cases of liver fluke.

It is distressing, Dr. Mani says, that in S. E. Asia—where people as a rule pay so much attention to personal cleanliness—they should have to live in such insanitary surroundings.

"Governments and municipal authorities", he observes, "certainly pay homage to the idea of improving local sanitation . . . . but when it comes to making adequate budget provision for clean water supplies, sewerage, drainage and garbage disposal, they put up a few more hospitals instead. And so the inexorable cycle of dirt and disease goes on."

WHO Assistance

During the period under review WHO has assisted governments in the SEA Region with 129 projects employing 238 WHO field workers, says the Report.

The Regional Office is handling annually a programme which amounts in value to about nine million dollars, inclusive of other extra-budgetary funds (these funds provide mostly for supplies and equipment furnished by UNICEF). The main fields of WHO assistance have been, as before, the control of communicable diseases, promotion of rural health services and extensive training programmes for all categories of health staff.

Malaria eradication programmes in the Region have made progress despite the difficulties the governments are facing, particularly with regard to finance. "Much greater need for adequate national staff, for better training and for stronger supervision at all levels was evident during the year", says Dr. Mani. *"Success in these programmes will depend primarily on the solution of the organisational, administrative and financial difficulties which at present exist."*

In the field of *tuberculosis*, WHO support in India is now being concentrated on helping to establish a well planned countrywide national tuberculosis programme. A pilot project was initiated in Thailand preparatory to the expansion of tuberculosis services throughout the country.

As a first step towards the launching of national *smallpox eradication* campaigns, some countries (Afghanistan, Indonesia and India) initiated pilot programmes which, it is hoped, will give answers to the many technical and administrative problems involved in the organisation of such campaigns.

• • • •

WHO assistance in the development of *epidemiological units* at national or state level was continued in both Ceylon and Indonesia. This type of activity, the value of which has now been demonstrated, will be further expanded in 1960 by starting similar assistance in Burma and by awarding fellowships for training state level epidemiologists in India.

Development of *rural health services* has been rather slow. This is due mainly to the shortage of staff available for a rapid expansion of services, to the unsatisfactory training of various categories of personnel and also to inadequate supervision, which reduces health centres to mere dispensaries.

As rural health centres develop, there is an increased need for referral services such as hospitals and laboratories. "Far too little has been done in the Region to develop public health laboratories" comments Dr. Mani, "and it is hoped to increase WHO assistance in this field".

In the field *of maternal and child health*, WHO and UNICEF continued to assist through urban or rural health centres and in developing the teaching of paediatrics and obstetrics.

WHO assistance in *environmental sanitation* has been concentrated, as before, on the education and training of sanitary engineers and sanitarians and on the environmental sanitation aspects of rural health programmes where pilot work has been promoted, e.g., in the state of Uttar Pradesh in India where house latrines in rural areas are becoming popular. In addition, survey of one urban sanitation project was started in the Greater Calcutta area. The Regional Office, through visits of advisers and consultations with the governments, has also started exploring ways and means to assist governments in the setting up of community water supply programmes.

Following the appointment of a Regional Adviser in Nutrition in the Regional Office, a review of the possibilities for WHO assistance in this subject has been undertaken.

Assistance to *medical education* continued by providing visiting professors and awarding fellowships. Direct assistance was given primarily in the teaching of non-clinical subjects and of preventive and social medicine.

At last a tendency is slowly developing in some countries towards closer *co-operation between the national health administrations and other ministerial departments*, e.g., the Ministry of Public Works for environmental sanitation, Ministry of Food and Agriculture for nutrition, Ministry of Education for health education, etc.

Health Highlights Country by Country

AFGHANISTAN :

The countrywide malaria eradication programme is in full swing. Training of personnel required for the programme is being carried out in the provinces as well as in the central malaria institute, Kabul. A total of 2·7 million people were protected by DDT spraying and anti-larval measures in 1959. The figure for 1960 was estimated to be 3·2 million.

A nation-wide campaign against smallpox, aimed at vaccinating the entire population within five years, has been adopted by the Government. Vaccine production within the country was increased to more than two million doses.

A Nursing Affairs Committee has been created by the Government to pave the way for a unified nursing policy for the country and standardisation of procedures.

A new 40-bed women's hospital at Kandahar has been completed as part of the government's programme to expand hospital facilities.

The country's first course in school health education for teachers was conducted at a teacher-training school. A national advisory council on health education has been set up by the government.

Orientation courses and field practice in rural health were organised for medical officers, medical students, sanitarians, auxiliary nurse-midwives and rural development workers.

BURMA :

Eight new hospitals have been constructed by the Government and two, built earlier, have come into operation. A 200-bed hospital is under construction in Taunggyi.

A new system of birth and death registration for the whole country, recommended by WHO, has, as a first step, been introduced in Rangoon and Insein.

As against the target of 9·55 million people, about 10·13 million were protected against malaria in 1959. The experiment of utilising voluntary labour in spraying operations has proved unsatisfactory and reorganisation is taking place.

As part of the rural health scheme 49 more rural health centres have been opened.

CEYLON :

Smallpox, cholera and plague are completely under control and malaria has reached a near-eradication stage.

As the incidence of typhoid fever, dysenteries and other gastrointestinal infections continues to be high the National Planning Council has given high priority to environmental sanitation and community water supply programmes.

Good progress has been recorded in the special campaigns against tuberculosis, venereal diseases and leprosy.

Under a phased programme for additional hospital beds, a new 300-bed eye hospital in Colombo is nearing completion. To oversome the congestion in the larger central hospitals, steps are being taken to provide the provincial and base hospitals with various specialised and laboratory services.

There are plans to set up Ceylon's second medical college in Kandy and a third near Colombo. The admission capacity of the Colombo Medical College and of the nurse-training schools has been increased.

INDIA :

A health survey and planning committee has been set up by the Government to review progress in health services and make recommendations for the future.

The full strength of the spraying units under the malaria eradication programme (230 endemic and 156·75 hypo-endemic units) is now in operation. Each of these is expected to cover a population of one million. In the first cycle of spraying in 1959 the total coverage was reported to be 93 per cent. The deficiencies noticed in the previous year's spraying programme have been removed in 1959-60.

The national tuberculosis programme, of which the National Tuberculosis Institute forms an important part, has made progress. A pilot study on BCG and another on the prevalence of infection among the 0-4 age group children were carried out.

The recommendation of the central expert committee on smallpox and cholera for a three-year smallpox eradication campaign has been accepted by the government.

INDONESIA :

A new scheme for the control of tuberculosis on the basis of home treatment has been accepted in principle.

The yaws programme continues to make headway despite a slowing down of work owing to shortage of funds. With the acquisition of a motor vessel, the Government has been able to extend anti-yaws activities in the outer islands.

Reorganisation of the health administration has been carried out with a view to making the health services more effective.

The training and reference centre for rural health in Bekasi has begun work. Rural health training areas are being set up at provincial centres.

The national malaria eradication services has been reorganised and the original programme modified after examining the staff and facilities available in the country.

NEPAL :

Health plans have received a larger budget allocation and a higher priority.

Nepal's first Maternity Hospital and Child Welfare Centre has been opened. This will also be used as a practice field for student nurses and midwives.

Under plans to develop health services in rural areas, 16 out of the 54 rural health centres scheduled to be opened, have already started work.

Several new sections have been created in the Directorate of Health Services as part of its reorganisation. Plans to create a regular cadre of medical officers and nurses in the national health services are under way.

Malaria eradication activities continued with emphasis on training of local staff, geographic surveys and spraying operations.

THAILAND :

Provision has been made in the country's six-year development plan (1960-65) for special attention to be given to those aspects of health which have a direct bearing on the economic life of the people (e.g., the control of malaria, T.B., yaws etc.).

The national malaria eradication programme is being continued with assistance from the U.S. International Co-operation Administration. A WHO advisory team made an appraisal of the programme in certain areas and its findings were sent to the Government.

In the yaws control campaign the field workers are gradually undertaking wider public health activities such as vaccination, inoculation, leprosy case finding, and treatment of minor ailments. The first yaws workers' training course on leprosy diagnosis and reporting was successfully completed.

A TB pilot project has been started, with assistance from WHO and UNICEF, in Bangkok. Comparative tuberculin studies and test vaccinations with BCG were carried out. The vaccine prepared by the Bangkok laboratory has been found to be of good quality.

Twenty-two second class health centres have been upgraded by the Government and 182 new midwifery centres opened. Land is being acquired to open a third midwifery school at Khon Kaen.

Leprosy control operations have been extended to five more provinces bringing a total of nine provinces in the country under the programme. From October 1955 to March 1960, 27,092 patients had been brought under treatment. The total cost (Government+WHO+UNICEF) of leprosy control in the pilot area (Khon Kaen province) is estimated at $4 a year per patient.

NOTES AND NEWS

The World Health Organisation

The annual report of the Director-General of the World Health Organisation for 1959 shows the wide range of the assistance given by the Organisation to countries throughout the world, both directly in the form of projects adapted to particular needs, and through international technical services of the more traditional type.

The 'project list', occupying more than half the volume, gives particulars of the projects, numbering more than 500, in which the World Health Organisation has been co-operating with the Governments of some 140 countries, sometimes in conjunction with other organisations. A summary narrative for each project states its aim, the nature of the assistance and progress made during the year, and adds, where possible, an assessement of the results obtained. The list, grouped by country and subject title under each of the six regions of the World Health Organisation, includes inter-country and inter-regional projects in which help is given to groups of countries working together on common problems—a type of project that appears to be increasing in importance.

In his introduction, Dr. M. G. Candau, director-general of the Organisation, directs attention to the fact that in 1959 the Organisation's work was carried on "against a general background of reduced tension in international affairs" and of "substantial improvement of economic conditions throughout the world, particularly in Europe"; and he finds growing evidence of the determination of the world leaders to devote more of their countries' resources to help nations still in the early steps of technological and economic development to raise their standards of living.

In discussing some of the changes of emphasis in the Organisation's programme, the director-general points out that communicable diseases remain one of the world's most serious health problems. For some, more knowledge is required; for others, such as malaria and smallpox, the methods are broadly known and what is wanted now is organisation, trained staff and money.

Among diseases calling for increased vigilance are the treponematoses. More than 100 million people still live in low-prevalence yaws areas, in several countries there has been a disturbing recrudescence of venereal syphilis, and a growing incidence of gonorrhoea has been found in 15 out of 22 countries surveyed. Investigations are being pursued in order to ensure that the most effec-

tive assistance can be given to countries combating these problems.

With tuberculosis, recent findings which show that domiciliary chemotherapy may for most cases be as efficacious as treatment in sanatoria are of particular importance to the many countries where there is an acute shortage of sanatoria beds.

Mention is also made of the newer developments against leprosy, bilharziasis, poliomyelitis and zoonoses, and of preparatory work with the view of eradicating smallpox.

Much preparatory work has been done during the year for the expansion and intensification of the medical research programme. Main targets and priorities, established with the guidance of the Advisory Committee on Medical Research, include the communicable diseases (with emphasis on viruses and diseases prevalent in tropical countries); for the highly industrialised countries, the chronic diseases, particularly cancer and cardiovascular troubles; nutrition, and problems of the increased risk of exposure to ionising radiations. The 'service to research' will include standardisation of nomenclature techniques and equipment, and the expansoon of, the Organisation's system of international reference centres.

Emphasis is also being given to the vast programme for the improvement of water supplies for communities, which is a primary objective in the attack on the enormous problems of environmental sanitation. The programme will be carried out in collaboration with departments of public works and with public administrations and financial authorities.

Annexes to the report give details of the membership of the Organisation, the main heads of the budget for 1959; and particulars of the secretariat structure and composition.

Training of Paramedical Personnel

The subject of the technical discussions at the thirteenth session of the WHO Regional Committee for South-East Asia, which will be held in Indonesia later this year, will be "Evaluation of training programmes for paramedical personnel". The training and use of auxiliary personnel has become an urgent problem in all countries of the Region, where the demand for health services is rising, particularly in rural areas, and the supply of professional health workers is inadequate. To help in the preparations for these discussions, Dr. N. R. E. Fendall is spending five months in the Region, studying available information on the subject and visiting the various countries concerned.

Dr. Fendall, who comes from New Zealand, is Assistant Director of Medical Services (Health) in Kenya, and a member of the Overseas Civil Service of the United Kingdom.

Indian Doctors in the Soviet Union

On August 26, the audience hall of Moscow's Friendship Club was filled to capacity. Moscow medical workers were playing host to their guests—a delegation of Indian doctors headed by Dr. H. N. Shivapuri, President of the Indian Medical Association.

The meeting was opened by Tatyana Zuyeva, Vice-President of the Union of Soviet Societies for Friendship with Foreign Countries. In her warm speech of welcome she expressed her happiness at the fact that Indian doctors were the guests of their colleagues in the Soviet Union. She expressed the confidence that the Indian doctors would utilise their stay in the Soviet Union for a thorough study of the achievements of Soviet medicine.

Professor B. Petrov, Vice-President of the medical section of the U.S.S.R.-India Friendship Society, introduced the Indian guests to the Soviet medical workers present in the hall, among whom were Mr. Tarasov, Director of the Sklifassovsky Institute, Prof. V. Chaklin, of the Central Institute of Traumatology and Othropaedics, Prof. M. Alexandrov, an eminent Soviet gynaecologist, and Prof. P. Androsov, a director of the surgical clinic of the Sklifassovsky Institute, who took part in developing a new vessel-suturing instrument.

Prof. Petrov stressed the profound interest shown by Soviet medical workers in expanding links with their Indian colleagues and exchanging scientific ideas to the benefit of the Soviet and Indian peoples.

In their speeches in reply, Dr. Tandon, Dr. Jalota and Dr. S. M. Sen thanked their Soviet colleagues for their warm feelings of friendship and understanding and noted that now, when the Soviet Union was helping the Indian people in building a number of pharmaceutical factories, the links between them were growing stronger than ever. Dr. Shivapuri remarked that the importance of the visit of Indian medical workers to the Soviet Union lay not only in the fact that they would have great opportunities to exchange experience, but also because it symbolised the unswerving desire of the peace-loving peoples of India and the Soviet Union for world peace.

On behalf of the Indian Medical Association, Dr. Shivapuri invited a delegation of Soviet medical workers to visit India in December to attend a conference of the Association.

Leprosy Relief Work

A successful function was organised in Berne by the Indian Ambassador, Mr. M. K. Vellodi on August 18, to educate the people of Switzerland in matters relating to leprosy and leprosy relief in India. The chief speaker on this occasion was Prof. Brand, who has been intimately connected with this kind of work at the Christian Medical College, Vellore, for the last 14 years.

Prof. Brand gave a talk, and illustrated it by a beautifully produced film, of which he himself is the author. The audience consisted of several distinguished surgeons, orthopaedists, social workers, representatives of the Government of Switzerland, and various other distinguished citizens of Berne. On this occasion the President of the Society announced that the Society had decided to donate on hundred thousand francs to the Christian Medical College, Vellore, for work connected with leprosy. The meeting was also attended by Dr. J. Ross Innes, Medical Secretary of the British Leprosy Relief Association, who had specially come to

Berne from London on the invitation of the Indian Ambassador. Both the talk and the film were very much appreciated, and it may be said that, for the first time, a serious effort has successfully been made in Switzerland to educate the people in matters relating to leprosy and leprosy relief in India.

Control of Leprosy in Andhra

With a view to controlling leprosy in the State, the Government of Andhra Pradesh have established 15 control units, with assistance from the Union Ministry of Health to undertake survey and treatment in endemic areas. Each centre covers a population of about sixty thousand and gives domiciliary treatment by administering sulpha tablets to persons suffering from the disease.

Besides, a training centre has been started for training medical personnel and doctors for the leprosy control campaign at Tirupati. The Gandhi Memorial Leprosy Foundation have started a training centre at Chikalapalli, near Bobbili.

Chest Disease Congress to Meet in New Delhi

The International Congress on Chest Diseases has accepted India's invitation to hold its seventh session in New Delhi. It is likely that the Delhi session will be held in January 1963.

Dr. R. Vishwanathan, Director and Professor of the V. P. Chest Institute, Delhi University, who participated in the sixth international congress in Vienna, forwarded the invitation.

Talks on Hereditary and Aberrant Cancer

Speaking on "Origin and Growth of Cancer" at Bose Institute, Calcutta, on 7-9-60, Dr. P. C. Koller, Professor of Cytogenetics, University of London, differentiated between cancer to which hereditary factors might have contributed and that which was presumed to have been mainly due to aberrations of the chromosomal structure.

With the progress of civilisation, he said, the occurrence of cancer appeared to have been increasing. In Britain the incidence of the disease had greatly risen. He referred to the possibility of a greater incidence of lung cancer among cigarette smokers.

Dr. Koller thought that the higher rate of multiplication of cells in cancerous tissues indicated that from simple growth the rate of growth of cells in such tissues was changed to that in a bacteria or yeast cell.

He illustrated with slides the effect of radiation on normal and tumour cells. He also discussed the effect of chemical agents, called chemical mutagens, on cells and heredity. The mustard gas used in World War I by Germans was shown to have produced a change in the chromosomal and nuclear structure of cells. Mutation was also produced in the fruitfly, Dresophils.

Probably, cancer-producing factors were mostly to be found in charge within the cellular structure, such as chromosomal aberrations, Dr. Koller said.

Measures to Ensure Drugs' Quality

The Lok Sabha on 6-9-60 passed the Drugs (amendment) Bill which empowers the Central Government to control the manufacture of drugs and to appoint inspectors and analysts to ensure the quality of the drugs.

The Bill, which has already been passed by the Rajya Sabha, was brought forward on the recommendations of the Pharmaceutical Enquiry Committee that the drugs standard control, which is being exercised by the State Governments, should be centralised for better enforcement. The Bill provides also a minimum punishment of one year's imprisonment and fine for the manufacture and sale of certain misbranded drugs.

Cholera Vaccine by New Method

Cholera vaccine is proposed to be produced in 'fermentors' at the Haffkine Institute in Bombay. The fermentor-method of production is a process designed to facilitate continuous 'tapping' of vaccines whenever required in large quantities at short notice. The fermentor method, which is in use in some antibiotic industries, is expected to help the Haffkine Institute to discharge its obligation of quick supply with greater ease.

At present the Institute follows what is known as the 'Haffkine flask' method for producing vaccines. It is a laborious process requiring an endless array of small culture-flasks to be kept constantly ready for use whenever necessary. Fermentors, when pressed into service, will ensure a steady and continuous supply of large quantities of the requisite vaccines which can be 'tapped' into ampoules, sealed and packed quickly. For a pilot project, the Institute has designed a 20-gallon-capacity fermentor and got it locally fabricated. When the scheme for the switch over from flasks to fermentors is fully implemented, in about a year, the Institute will have large fermentors of about 100-gallon capacity each.

The fermentor-method is expected to reduce the cost of production as it eliminates, among other factors, the need for separate sterilisation and distillation processes from individual flasks. At present cholera vaccine costs Rs. 1·25 nP. for a vial of ten doses. It will also reduce the production period from three weeks to one week.

Vaccines for typhoid and plague can also be manufactured in fermentors. But this is not considered necessary as the demand for them is not very great.

Rs. 2-Lakh Scheme for Anti-tuberculosis Project in Delhi

The Standing Committee of the Delhi Municipal Corporation recently approved a scheme prepared by the Commissioner for the treatment of tuberculosis patients. It sanctioned the creation of 12 posts of medical officers, health visitors and other officials for six months. The expenditure by way of wages, medicines and an x-ray unit will be about Rs. 2 lakhs.

The Commissioner suggested that as a first step, the Corporation might introduce a domiciliary treatment scheme in a specified area where the incidence of tuberculosis was high.

On the basis of available data, the area bounded by Kutab Road from Teliwara to the Kirori Mal School

site on the east, Bahadurgarh Road up to the municipal dispensary on the north, M. M. Road from the dispensary to Idgah Road crossing on the west and Idgah Road to Kirori Mal School on the south was considered most suitable for introducing the scheme. The estimated population of the area is 1·20 lakhs.

The Commissioner's report said that an estimated 20,000 people in Delhi were suffering from tuberculosis and half of them were in an infective stage. The mortality rate in 1948-1957 had decreased from 0·5 per 1,000 to 0·2 per 1,000 but the incidence rose from 2·7 per 1,000 to 4·7 per 1,000.

At present two hospitals and six clinics, aided by 26 health visitors, were functioning in Delhi while the total bed strength at the hospitals was 1,213. In 1958-59, 984 patients were admitted at the S. J. T. B. Hospital and 624 at the T. B. Hospital in Mehrauli. The number of cases recorded at the clinics was 13,799.

CORRESPONDENCE

The Editor is not responsible for the views expressed by correspondents.

Treatment of Infective Hepatitis

Sir,—Please allow me to comment on the above article by Prof. P. N. Laha in the Practitioners' Series published in your issue dated July 16,1960, pp. 54-57. It gives a good account of the theory about this disease but little practical guidance in its management.

Under diet, figures in grams and total calories are given without any detail of how and from which items of commonly available materials, these should be obtained. In this matter the general practitioners would welcome practical suggestions like, 1 cupful of curds, 2 eggs, ¼ seer of milk, etc., giving their approximate protein and calorie content. It is difficult for general practitioners to refer to various textbooks for tables of calories and work out details of a diet. The articles in the Practitioners' Series should preferably provide these details.

Under prevention, the dose of gamma globulin is given. It is my impression that gamma globulin is not commercially available in India. The writer should be specific about availability of drugs and reagents recommended.

Under hepatic coma—complication, the use of oral neomycin is recommended. This drug is not advertised in India and I presume that it is not available here. Similarly, in hepatic coma the use of sodium glutamate is advocated without mentioning whether it is to be given orally or parenterally.

The source and the name of manufacturer should preferably be stated in case of uncommon drugs although it may raise issues of medical ethics and journalistic etiquette.

An article in the Practitioners' Series should be specially prepared taking into account the requirements of general practitioners and should contain as much of practical details as possible. If contributors kindly bear these points in view, I am sure, they will readily provide the needed information. In this way the value of these articles will increase and their popularity will be enhanced.

Ahmednagar. D. B. LELE

* * * *

With reference to the comments by Dr. D. B. Lele on my article, Treatment of Infective Hepatitis (*J. Indian M. A.*, **35**: 54, 1960), my answers point by point are:

1. Food habits differ widely, depending on personal habits, likes and dislikes, social and economic conditions, etc. As such it will be difficult to chalk out one or two scheduled diet charts for a patient of infective hepatitis where anorexia is an important symptom. Hence in my article, under diet, figures in grams have been detailed. Such a procedure is scientific and would also help a physician to prescribe diet for his patients of infective hepatitis depending upon, as said above, the habits, likes and dislikes, and other factors which may govern their (patients') diet. It is presumed that every physician possesses a basic knowledge of dietetics. He can always refer to any book or literature dealing with diet, if so required. I do not think that it would create a very difficult problem for a practitioner.

2. It is true, that gamma globulin prescribed for prevention of infective hepatitis is not freely available at present. But that does not warrant negation of its knowledge.

3. Wide-spectrum antibiotics have been recommended for the treatment of hepatic coma due to reasons as detailed in the article. Any one of them can be used. But neomycin, having certain advantages over other antibiotics, have been mentioned in the article. Depending upon its availability and cost it would be the antibiotic of choice.

4. Sodium glutamate is to be administered intravenously.

In the end, I have to state that I am glad to note the comments about the article. More such comments would help writers to know the viewpoints of their readers and thereby there would be better orientation of articles in Practitioners' Series.

Gwalior. P. N. LAHA

Plight of Medical Officers in S.S.R.C./A.M.C.

SIR,—The Short Service Regular Commission in the Army Medical Corps was reintroduced in the post-Independence era in 1948 with a tenure of 3 years' service extendable up to 5 years. This term was further extended by another two years, i.e., altogether to 7 years in 1953. Whereas all the other branches and arms of the Army have been making their irregular officers regular by abolishing Temporary Commission and Short Service Regular Commission, why the medical branches are still recruiting on short term basis is incomprehensible. It is equally difficult to understand why they have not yet absorbed all the S.S.R.C. Medical Officers and taken them into the permanent cadre

by abolishing the S.S.R.C. Actually the Medical Directorate is maintaining three standards: (1) One for permanent regular Commission, i.e., 8 years of service—Major; (2) Emergency Commission, i.e., 10 years of Service—Major; (3) S.S.R.C.—No Majorship, whatever the period of service may be.

In 1952/53 three-fourths of the total medical officers belonged to the cadre S.S.R.C. They were given most hazardous and arduous tasks under trying conditions and had to look after the health of all Defence personnel including the Army, the Navy and the Air Force. It is a fact that the Medical Directorate has created five top-ranking posts, viz., two Major Generals and three Brigadiers in the A.M.C. with the help of the numerical strength of these S.S.R.C. Medical Officers. In spite of all, these Medical Officers are thrown out in the wilderness of unemployment without even an adequate notice. There are cases on record where they have been released from the Army on two days' notice. In fact the S.S.R.C. Medical Officers are the most neglected of all officers in the Army. Nobody cares to look after their welfare. They have been exploited to the furthest extent. It is high time that this S.S.R.C. for Medical Officers should be abolished altogether. The Army is not deriving any benefit from this type of service, neither is it doing any good to these ill-fated doctors. I am, etc.

Calcutta. S. S. BAGCHI

At the All-India Medical Conference held at Indore in December 1959 a resolution was passed to stop Short Service Commission. The resolution is quoted below:

Resolution No. 17—This Conference notes with concern that a large number of Short Service Commissioned Officers in the Armed Forces Medical Service are thrown out of service sometime after 6 years or more. This is detrimental to the interest of the medical personnel concerned and to the recruitment in emergency. This Conference urges upon the Government to absorb them permanently.

—ED., *J. Indian M. A.*

REVIEWS

Childbirth without Fear—By Grantly Dick Read. 4th edition, 1960; William Heinemann Medical Books, Ltd., 15-16 Queen's Street, Mayfair, London W. 1; 9⅛" × 5⅝"; pp. xvi+266; price 12s. 6d. net.

The book is the outcome of the life-long work of the author as described in his preface to the fourth and the first edition. This is a work based on practice and clinical observation in practical obstetrics, as claimed by the author. The author has written exhaustively to establish his theory of 'fear-tension-pain syndrome'. According to him, fear is the cause of all evils particularly pain in natural childbirth.

In Chapter I, the author reviews some of the historical facts concerned with the evolution and progress in the science of obstetrics. Further progress should be made and with this view in mind he endeavours to write the subsequent chapters of the books.

In Chapter II, motherhood has been discussed from several points of view. The author deplores that motherhood has not received the consideration it deserves. He stresses the importance of natural childbirth without fear and of practice to that effect. According to the author fear-tension-pain syndrome should be understood and on the basis of this attempts should be made to allay the prospective mother's fear from the very first visit.

In Chapter III, the author describes this theory of natural childbirth. The woman in Whitechapel—whose labour the author had to attend about 30 years ago, and who refused to have the mask of chloroform, made a revelation to him. "It didn't hurt. It wasn't meant to, was it doctor?" He believed that superstition, civilisation and culture influences the mind of the women and are responsible for the fear and anxieties of labour. According to him, fear, tension and pain are three evils opposed to the natural childbirth. They have been introduced in course of civilisation by the ignorance of those responsible for the management of childbirth.

The anatomy and physiology of uterus, ovum, etc., have also been touched. He admits that the physiological principles assigned to natural childbirth are not without dispute, but his comment on the physiology of childbirth has been made to clarify the object of the book.

In Chapter V, the cause of pain in labour has been explained. The biological purpose of pain is protective and no physiological function in the body gives rise to pain in the normal course of health. How is it that normal childbirth should so consistently be associated with pain? Every effort has been made to impress on the reader how fear plays the most important contributory factor in the causation of pain in otherwise normal labour.

In Chapter VI, factors lowering the pain threshold have been discussed. Anaemia, physical tiredness, mental worries are important. Depressions and disappointments are potent pain intensifier. Autosuggestion plays an important role in pain production in labour. Impressions stored up in the memory of past experiences may have similar effect. The author maintains that no normal labour in a well trained mother should give rise to any discomfort necessitating anaesthesia.

In Chapter VII and in subsequent ones the author explains the importance of fear in the production of pain. The influence of the emotional factor in pregnancy and parturition is now recognised. What is fear? An emotion arising from the primary instinct of flight. It is a natural protective agent which enables one to escape from danger; when danger is avoided there is no fear. He explains how from knowledge and training confidence may be brought in to eliminate fear. Apart from apprehension and doubt, every pregnant woman is subjected to fear-producing factors peculiar to pregnancy and childbirth, e.g., from early age the girls become aware that childbirth is pain-producing and is not without danger. The author explains how circumstances, descriptions in serious books and fiction add to it.

In Chapter X, the author dilates on the effects of fear. How the expectant mother has been made to believe that she is to suffer from pain even if everything goes well. The author feels that the general teach-

ing in antenatal clinics is primarily scientific, only routine work is done and sufficient attention is not given to the emotional aspects to allay fear. The author rejoices in that this aspect is being appreciated more and more now-a-days. The diet in pregnancy has also been described and discussed.

In Chapter XII, the phenomenon of labour has been described. How the author started to believe "Tense women, tense cervix." Experience and practice helped him to introduce the practice of relaxation in pregnant women with beneficial result in producing painless labour. He advises the practice of relaxation to be started during the last four or five months of pregnancy.

In Chapter XIII, relief of pain in labour has been described. According to him, it depends on proper understanding of the fear-tension-pain syndrome. Fear inhibits the normal sequence of events during labour. He believes that only those cases where the woman has not been prepared for childbirth—what he calls cultural labour—requires help for relieving pain.

The author warns that his method must not be misunderstood for hypnosis.

In Chapter XV, details of his method of natural childbirth have been described for general understanding. The general principles of good obstetrics must be recognised. In addition, he discusses the conduct of labour, from the point of view of the woman herself, her mental condition and changing emotional state, and describes his method of obtaining physical and mental relaxation by antenatal education.

In subsequent chapters the importance of education of this natural process to women has been stressed, and breast feeding with its effects on the mother has been discussed. Detailed description of antenatal procedures and methods of preparation for labour have been included. There are photographs to illustrate the relaxing method and preliminary relaxation.

The book is well bound. It should be read not only by men in the profession but also by pregnant mothers and women in general as intended by the author.

D. GHOSE

Surgery and Clinical Pathology in the Tropics—By Charles Bowesman, O.B.E., B.A., M.D., F.R.C.S.E., F.A.C.S., D.T.M. & H. (ED.). 1960; E. & S. Livingstone Ltd., Edinburgh & London; 10" x 7"; pp. viii+1068; price £5. 10s. Regd. postage abroad 6s.

The book is a record of materials collected in various parts of West Africa. It is a voluminous book comprising of 1050 pages of text divided into 30 chapters. There are 321 illustrations.

There is a vast amount of information including that related to customs of different tribes and their indigenous ingenuity.

It does not confine itself strictly to 'surgery' and gives meticulous details of surgical, medical and laboratory procedures.

The author advocates 'basal circumcision' for elephantiasis penis by retaining 'all the inner skin of the prepuce' and 'retracting (this) back to cover shaft of penis'. In the reviewer's experience such cases come back with swelling of this retained skin and require a second operation.

Written evidently for general medical officers, it will certainly be of great help to the lone worker working amongst underdeveloped people. It would have been more easily assimilable if it were less discursive and verbose.

The get-up is excellent.

S. D.

Biology of the Pleuropneumonialike Organisms, *Annals of the New York Academy of Sciences,* Volume 79, Art. 10, January 15, 1960—Editor-in-Chief, Otto V. St. Whitelock; 9" x 6", pp. 454; price $5·00.

The monograph consists of a series of papers presented for the Conference and Biology of the Pleuropneumonialike Organisms held by the New York Academy of Sciences. The papers have been adequately classified into seven parts comprising Morphology and Classification, Isolation and Propagation, Classification of PPLO, Physiology, Serology and Chemotherapy, Animal Pathology and Human Pathology. It is a very comprehensive account of all the advancement of knowledge which has accumulated since last several years from intensive research on the PPLO group of organisms. Though naturally a very specialised monograph it should certainly be useful for those who are interested in the advancement of bacteriology.

D. C. LAHIRI

CORRIGENDUM

It is regretted that the legends appearing with the photographs on page 333 of October 1, 1960 issue were inadvertently interchanged during printing. The photographs are reproduced here with the correct legends.

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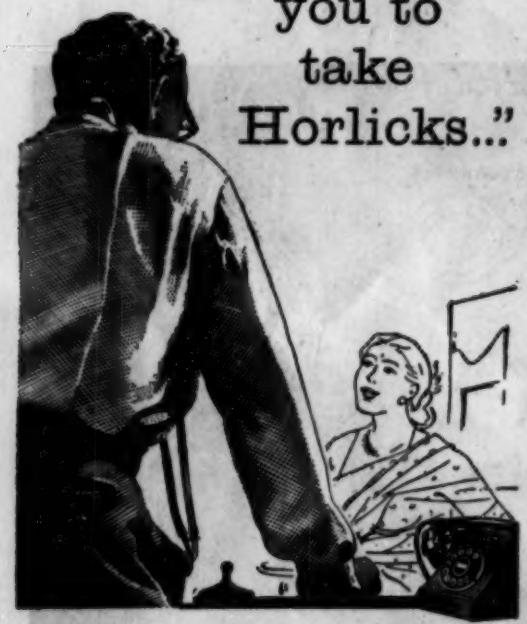

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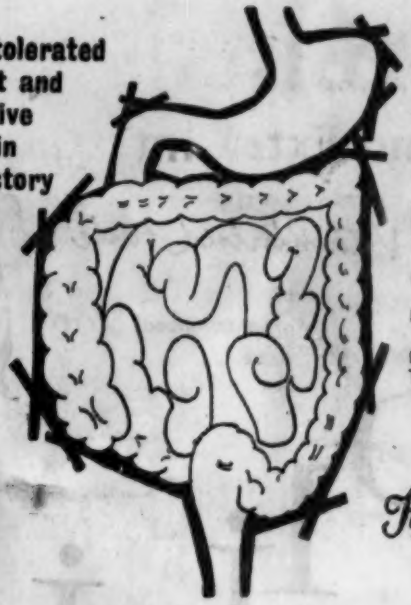

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K. N. Gour, M.D., F.R.C.P.E., F.R.F.P.S.G., D.C.H. (Eng.), D.P.H., and Sudhir Gupta, B.Sc., M.B.,B.S., D.C.H., *Current Medical Practice*, 1959, 3, 135.

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Amebiasis may be asymptomatic or may produce symptoms indicating low grade infection, inflammatory colonic disease with or without diarrhea, or symptoms resembling appendicitis, gallbladder disease, or peptic ulcer.[1]

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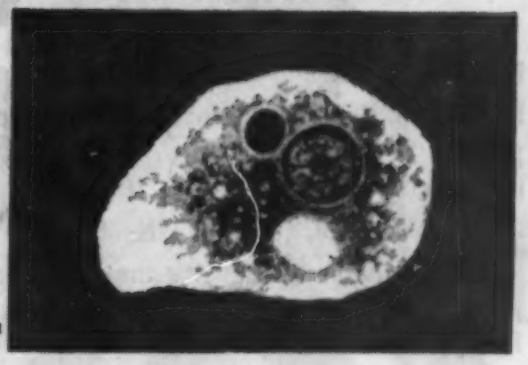

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3 Conan, N. J. : *Am. Jour. Med., 6 : 309*, Mar., 1949

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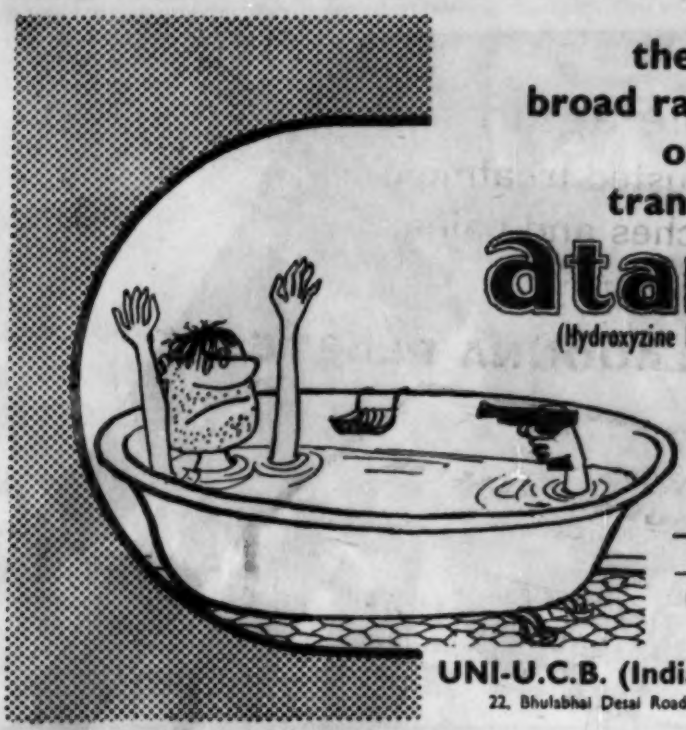

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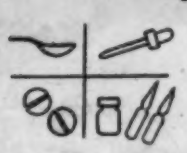

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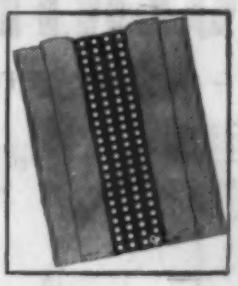

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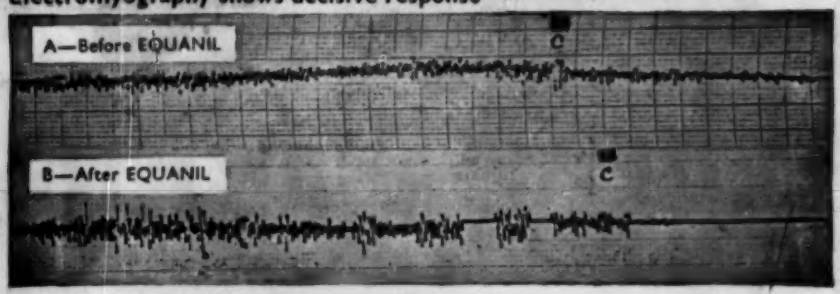

Tracing A, made before medication, reveals a marked action potential following the command to relax at point C. Note the difficulty and slowness with which the patient responded.

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1. Dickel, H. A., et al.: West. J. Surg., April, 1956.

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in syrup base.

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Benzyl Alcohol 2%
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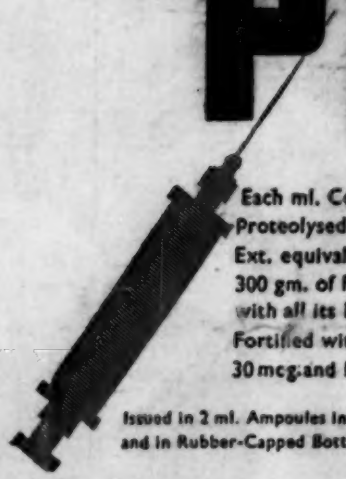

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